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Volume 6 August, 1953 Number 1

CONTENTS*

Aseptic Necrosis of the Acromial End of the Clavicle. Presentation of Four Cases.
Eugene R. Kraus, D.O., F.A.O.C.R. 3

Acute Abdominal Conditions. Roentgen Diagnosis.
A. H. Witthohn, D.O., Grand Rapids, Mich. 6

Osseous Manifestations of Systemic Diseases in Infancy and Childhood.
Hervey S. Scott, D.O., and Harve J. Helton, D.O., Kansas City, Mo. 8

Differential Diagnosis of Pulmonary Neoplasms.
Paul Bramnick, D.O., Flint, Mich. 9

Radiographic Diagnosis of Urinary Tract Neoplasms in Infants and Children.
Robert R. Rosenbaum, D.O., New York City.....13

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Aseptic Necrosis of the Acromial End of the Clavicle

Presentation of Four Cases*

EUGENE R. KRAUS, D.O., F.A.O.C.R.

The human shoulder is perhaps the most fascinating joint structure in the body. Codman[1] spent a professional lifetime studying its anatomy, function, and pathology. There is hardly a physical act which does not involve some motion at the shoulder, but that so little disorder results from so much use attests to its marvelous design and structure.

Study of the comparative anatomy of the shoulder turns up some conflicting evidence for the need and importance of what Codman[1] calls, "one of man's greatest skeletal inheritances"—the clavicle. "We are proud," he says, "that our brains are more developed than those of animals; we might also boast of our clavicles."

In birds, the strong lateral thrust of the wings (forelegs) necessitates a sturdy bridge across the anterior thorax, and thus the clavicles have fused to form the keel or wishbone, a nice adaptation of form to function. In fact, all flying and climbing animals have clavicles except bears. All animals which use the forelegs for clawing and prehension have clavicles, excepting a group of large carnivores such as the lions, tigers, leopards, and domestic cats whose rudimentary clavicles are suspended in the muscles and do not articulate with either the sternum or scapula. Animals which specialize in running, grazing, and swimming do not have clavicles; the beaver, which uses its forelegs in building huts and constructing dams, has the best developed clavicle of all; seals, whales, dogs, foxes, wolves, horses, cows, deer, pigs, and hooved animals have none. In man, the normal, manifold activities demanded of the shoulder call for a clavicle.[1]

There is frequent occasion to refer to the effect of man's upright posture on the subsequent development of his bony skeleton. Reference to Wolff's law will also show the inseparability of function and anatomy. Whereas in quadrupeds the foreleg helps support the body against the pull of gravity like the pier of a bridge, in man only the muscles counteract the pull of gravity on the upper extremity, and no part of the body is supported by the bones of the shoulder. The humerus, which is not regarded as part of the shoulder girdle, hangs suspended like a pendulum from the joint formed by the clavicle and scapula. Strictly speaking, the girdle is formed by both clavicles and scapulae, deficient in the back where the scapulae do not meet, and completed in the front by the sternum.

The clavicle, whose importance is so conflicting, acts as a fulcrum for the muscles used to give lateral motion to the arm. It is a *strut* connecting the rest of the shoulder with the skeleton and prevents the upper portion of the shoulder from approximating the body, as is the case in cleidocranial dysostosis. It is capable of limited circumduction facilitated by a double joint at the sternal end. In motion, it acts like the boom of a derrick, enabling the trapezius to raise the whole shoulder.

According to Gray,[2] the articulation between the clavicle and sternum is a double arthrodial joint, with the articular surface of the clavicular side being much larger than the sternal side. The articular cartilage is thick and an articular disk separates the joint into two chambers. It has a limited range of motion in nearly every direction: upward, downward, backward, forward, and in circumduction. When these motions occur, the clavicle carries the scapula with it, gliding the latter over the outer surface of the chest. Codman[1] adds that the sternoclavicular joint is the only point at which the weight and power of the shoulder get direct support from the rest of the skeleton when pushing with the arm.

The acromioclavicular articulation, according to Gray,[2] is a single arthrodial joint with an inconstant articular disk. When it is present, it only partially separates the articular surfaces and occupies the upper part of the articulation. The joint has important functions in the movement of the upper extremity. The articular end of the clavicle glides on the acromial articulation. The scapula is permitted to rotate backward and forward on the clavicle. If there were no joint between the scapula and clavicle, circular movement of the scapula on the ribs, as in throwing the shoulder backward or forward, would be attended with a greater change in the direction of the shoulder than is consistent with the free use of the arms in such positions, and it would be impossible to give a blow directly forward with the full force of the arm.

Codman[1] describes the acromioclavicular joint as being very small in comparison to the thrust and power it has to transmit. After 25 years of study of the motions of the shoulder, he confessed that his curiosity on the subject had yet to be satisfied. The surfaces of the acromioclavicular joint slide a little, rotate a little, tip apart a little, and act a little like hinges. Even though the motion is so minimal, complete fusion at this point would oblige a very small structure to bear the stress for a whole extremity. The joint was therefore made to be slightly movable and not too rigid; otherwise it would be very much subject to injury. The range of motion is only that permitted by the coracoclavicular ligaments. The greatest weakness of this little joint, Codman[1] says, seems to be downward force applied to the acromion without touching the end of the clavicle. A blow or fall on the tip of the acromion without at the same time striking the clavicle commonly ruptures the acromioclavicular ligaments.

In summary, it would seem that rather free motion is possible at the sternoclavicular joint and that much stress must be transmitted through it since it is the one point at which the forces from the shoulder pass to the skeleton. One might expect injury and wear and tear to occur here more frequently than in the better protected, less movable acromioclavicular joint. Such lesions as the avascular necroses, which have been reported in weight-bearing bones and bones subjected to crush injuries, seem to occur only with great rarity at

*Dr. Kraus died shortly before the meeting of the American Osteopathic College of Radiology at Columbus, Ohio, October, 1952. His scheduled paper was presented in memoriam by Robert R. Rosenbaum, D.O.

the shoulder. In fact, to date only case in the sternoclavicular joint has been reported.

The purpose of this paper is twofold. First, it endeavors to discuss briefly the subject of avascular necrosis of bone, since it is of paramount significance to the orthopedic surgeon, the radiologist, and the general osteopathic physician. Secondly, it will present four cases of aseptic necrosis of the acromial end of the clavicle. To my knowledge no cases of this type have been reported in the literature. Despite the fact that avascular necrosis has been studied by the medical profession for years, there are great discrepancies in knowledge regarding its etiology and treatment. As I pointed out in a previous article,[3] whatever knowledge has been gained on this subject is cumulative and extends back as far as 1875 when Langer claimed that bone contained end-arteries. The next important event occurred in 1888 when Koenig first presented the subject of osteochondritis dissecans. In 1895 Roentgen discovered the x-ray and, since this revolutionized the study of bone, numerous areas of osteochondrosis and aseptic necrosis have since been described. The most frequently involved area is the femoral head in both children and adults. Other areas are the tarsal scaphoid, the head of the second metatarsal, the tibial tubercle, the femoral and humeral condyles, the semilunar, and the carpal scaphoid. The lumbar and dorsal spines are frequently involved in adolescents.

Aseptic necrosis and osteochondrosis may be divided into three groups. Group 1 involves adults, usually following trauma. Group 2 affects children and adolescents during the growing period. Group 3 includes those cases which are part of a systemic disease process.

In the article mentioned above,[3] I gave a concrete definition of aseptic necrosis and, since the thinking in that day followed the teaching of Axhausen, I pontifically stated that aseptic necrosis was death of bone caused by infarction. The present-day definition by Pugh[4] is as follows:

> . . . The term "osteochondrosis" is defined as "a disease of one or more of the growth or ossification centers in children which begins as a degeneration or necrosis followed by regeneration or calcification." . . . Some lesions included in this group are the direct result of trauma; this is true especially in adults where there are fractures which definitely disrupt the blood supply and in such cases the lesion should be called "aseptic necrosis." The term "osteochondrosis" is best used to describe those lesions that develop spontaneously or without definite evidence of gross trauma and these are found predominately in children or adolescents.

From the above definition it will be observed that the etiology of this condition is not distinctly understood, and it is now considered that in addition to the embolic process which is operative and causes loss of viability of the bone, there is some other factor present which predisposes to the process of infarction. This is evidenced by the fact that many of these cases have multiple areas of involvement and that in Perthes' disease about 20 per cent of cases involve both hips without any history of accident.

The cause of disease in the small bones, particularly in the wrist, and in the heads of long bones, can be definitely traced to trauma, so that there is no question of etiology in these cases. In caisson disease the cause is definitely known and therefore the pathologic physiology is well understood.

The pathologic process, as noted from the definition, is a disturbance of circulation, resulting in death of the active parts of bone, either in part or whole. Such active elements as osteoblasts, osteoclasts, marrow cells, and even trabeculae, disappear. Amorphous calcium is found in the soft tissue. The blood vessels are absent from the necrotic area. Depending on the length of the disease process, varying degrees of deformity may be present. The above description applies to the degenerative stage. Eventually regeneration occurs as a reaction of healthy bone to the irritation of a foreign body, the sequestrum. Granulation tissue forms and invades the dead bone. Accompanying this are osteoclasts which act as macrophages and help carry away considerable of the nonviable detritus. Osteoblastic activity is also begun and a new fibrous framework is laid down and eventually new bone replaces the inanimate tissue. If the bone involved is not in a weight-bearing area or if the patient has not been permitted to bear weight, residual deformity will not be present. In untreated patients who bear weight in dependent parts, deformity occurs.

The x-ray findings are the most important single element in making an early diagnosis of osteochondrosis or aseptic necrosis. The x-ray film follows the pathologic findings. If a case is seen in the early stages nothing may be observed. In suspected cases, therefore, re-examination must be made fairly soon. In children, usually the first indication of circulatory disturbance is an irregularity of the epiphyseal line and perhaps distortion of the capital epiphysis of the femur. With further degeneration areas of erosion which have a cystlike quality and areas of increased density which represent nonviable calcium are observed. Eventually there may be an almost complete disappearance of the head of the bone or the bone may become compressed and deformed. The appearance of the head is sometimes described as fragmented. After about 1½ to 3 years, evidence of repair will be noted. Areas of calcification will be seen; in the degenerated area the deformity will become less; a framework of bone will be laid down, and eventually complete reformation of the head and neck of the bone, with or without residual deformity, will occur, depending on many factors, such as proper treatment and the presence of a good anastomotic circulation. The importance of x-ray study in prognosis and treatment of bone death and repair can be seen.

The following cases are apparently examples of this same process in a location not previously reported to be involved.

Case 1. A 34-year-old osteopathic physician, an Army major, was first seen by me October 10, 1942. His chief complaint was pain in the left shoulder, which hurt him particularly in rainy weather and when performing any manual labor. He had a constant feeling of soreness of the shoulder, particularly on abduction and elevation.

On August 5, 1942, 2½ months before I saw him, while teaching commando tactics, he was thrown to the floor and struck his left shoulder. He had excruciating pain, and immediately went to Army headquarters where he was x-rayed and told to go home and rest. The pain in his shoulder persisted during the following week. On August 12, the patient received orders for foreign service. He was x-rayed again, relieved from duty, and hospitalized for a short time. He was advised that his injury was negligible. He was not, however, sent overseas. In October, 1942, he was rehospitalized, told to remain in this country and to report once more in January.

By this time the patient became quite concerned

with his condition and consulted me. X-ray studies showed a separation of the constituent portions of the acromioclavicular articulation of the left shoulder, a swelling of the soft tissue around the joint, and an erosion and irregularity of the acromial end of the clavicle. Since the eroded bone suggested several possibilities the case was discussed with other radiologists and with several orthopedists who inclined toward a diagnosis of regional tuberculosis. A chest film was negative, and the personal history was not suggestive of tuberculosis.

In January, 1943, the patient was again hospitalized, and the surgeon who previously thought the shoulder findings negligible and negative now favored a diagnosis of tuberculosis and wanted to discharge him from service. He was sent to an Army hospital where the orthopedic surgeon in charge said his condition was negative and that he should report back to duty. He did so, still having pain in his left shoulder. A year later he again received overseas orders but was deferred because of the appearance of the left shoulder on the x-ray studies. A medical corps general became interested and wanted to decide whether he should be in limited or general service. The medical board convened and declared that he should be put on limited service, and the orthopedist in charge diagnosed an aseptic necrosis of the acromial end of the clavicle.

Soon the patient was discharged from the service, and in 1945 returned to the general practice of osteopathy. He has had very little trouble with his shoulder and it bothers him only when he treats very heavy patients.

Films taken since 1946 demonstrate a persistent separation of the acromioclavicular articulation. They show complete repair of the eroded area with sclerosis of the margin of the acromial end of the clavicle. The right shoulder is normal in every particular.

This was my first experience with a case of aseptic necrosis of the acromial end of the clavicle, and this patient's experience illustrates the difficulty of making a diagnosis.

About this same time I made an x-ray study of a flier who had hurt his shoulder in an accident, and I found a similar involvement. The fact that two such cases appeared together made me conscious of an eroded end of the clavicle as a possible cause of shoulder pain.

Case 2. A 22-year-old actor, dancer, and singer was first seen on December 15, 1948. He gave the following history: While lifting his partner during a dance routine on December 11, he felt a pain in his right shoulder, the pain being localized in the acromioclavicular articulation. Four days later, when he appeared for treatment, the patient still had shoulder pain, but it bothered him only when he supinated his hand or lifted his dancing partner. He was still working but had discontinued that portion of the act.

The patient's history revealed a knee condition when he was about 14 years of age which had been diagnosed as Osgood-Schlätter disease. There were no other pertinent facts in his history.

Comparative x-ray studies of the shoulders showed a separation of the right acromioclavicular articulation, a swelling of the soft tissue of the right shoulder, and an area of erosion on the superior and external portion of the right clavicle where it participates in the acromioclavicular articulation. The left shoulder by comparison was smooth. The diagnosis was synovitis of the right acromioclavicular articulation, accompanied by aseptic necrosis of the distal portion of the clavicle where it forms part of the acromioclavicular joint.

Physiotherapy was prescribed and 2 months later, in February, 1949, the patient was x-rayed again. At that time he still had a painful shoulder, was still unable to lift his dancing partner, but otherwise felt no inconvenience. The x-ray studies showed the eroded area, but there was a slight calcification in the center of the nidus which was not observed in the previous study.

In May, 1950, the patient reported for another x-ray study. No treatment was given in the interim. He was still conscious of his shoulder, but whereas previously it had ached considerably he now felt no aching. At this time there was still a slight separation of the articulating ends of the joint as compared with the normal left shoulder, but there had been a periosteal overgrowth of bone and a gradual repair of the eroded area.

I would have liked to follow this case further, but the opportunity did not occur. The interesting feature is that, having seen several similar cases, I was able to recognize this entity when I met it. It is apparent that in cases of aseptic necrosis of the acromioclavicular articulation there is usually a history of trauma, it occurs in adults, and there is an associated synovitis and perhaps a destruction of some of the ligamentous tissue. In this particular case the x-ray studies showed very clearly the process of degeneration and slow repair, the so-called creeping substitution described by Phemister.[5]

Case 3. A ballet dancer and teacher, aged 45, gave a history of being constantly conscious of her right shoulder and of feeling one spot which was just "not right." This condition had persisted intermittently all winter. She had received manipulative treatment at irregular intervals, but because she had to travel, she could not follow any treatment consistently. Tenderness was noted throughout the upper humeral area, but point tenderness was elicited at the acromioclavicular joint.

X-ray studies of the right shoulder showed a circumscribed area of diminished density at the tip of the distal portion of the clavicle near its superior margin. The normal left shoulder showed a smooth clavicle without any defect. The diagnosis was aseptic necrosis of the acromial end of the right clavicle.

This case was chronic and apparently was in the second stage, the degenerative stage having been completed and the regenerative stage being present. New periosteal bone had formed and eventually the area of diminished density will be filled in.

Case 4. A man, aged 41, came to me with a chief complaint of pain in the right shoulder, especially in the acromioclavicular articulation. It had been about 3 years since he first noticed the condition, following serving at tennis. He had attempted to play tennis since then, but was unable to do so. Playing golf did not bother him. He had a tender area in the right acromioclavicular region. The patient also noticed pain when he hung up his coat and, at times, when he lifted a cup. He had recently consulted his osteopathic physician, but until then he had received no treatment.

X-ray studies of the right shoulder showed a swelling of the soft tissue, perhaps a slight separation of the acromioclavicular joint, and a distinctly eroded

area at the acromioclavicular margin of the right clavicle on its superior surface. Some periosteal new bone appeared to be present.

This case is of interest because it follows the pattern of the other cases. It seemed to be in the regenerative stage. Apparently the necrotic area was much more extensive in this case than in Cases 2 and 3, consequently the process of repair is taking much longer.

SUMMARY AND CONCLUSION

I have presented four cases of disease of the shoulder which could readily be confused with many disturbances and which are examples of an entity that has not, to my knowledge, previously been described in the literature. All four patients complained of shoulder pain brought on particularly by abduction and elevation of the shoulder. Pain persisted, but only on motion. The pain resembled that of other shoulder lesions, such as infectious disease, bursitis, tendonitis, or ruptured tendon. On careful physical examination point tenderness and pain were localized at the acromioclavicular joint. The determining factor in the diagnosis was the roentgen studies.

From these four cases I believe it has been established that aseptic necrosis is an entity and may be the cause of symptoms. Since there is a cycle of degeneration and repair, I think that the treatment may be expectant, consisting of watchful waiting and avoidance of motions which cause pain. If the response is not satisfactory, then surgery may be considered.

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Acute Abdominal Conditions

Roentgen Diagnosis

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In this presentation, an attempt will be made to portray the role of roentgenology in the diagnosis of acute abdominal disturbances which are considered as emergency or urgent conditions. Studies in these conditions are often omitted or overlooked either because of failure to appreciate the value of roentgen examination or hesitancy to submit the patient to the examination. The latter reason plays a major part where the x-ray department is not convenient to the bed area or to the admitting area, and the patient must be transported up or down stairs.

Because of the scope of the subject under discussion I shall consider mainly those diseases in which radiography without contrast material is often conclusive. The discussion will include biliary and urinary obstruction due to calculi, perforation of hollow viscera, intestinal obstruction, paralytic ileus, mesenteric thrombosis, pyloric obstruction, acute pancreatitis, extra-abdominal pathologies causing acute abdominal symptoms, and traumatic rupture of abdominal viscera.

In many cases, the findings obtained from plain radiography are sufficient to make a diagnosis. Indeed, the administration of contrast material is sometimes contraindicated. The patients must often be examined without special preparation, since they are brought to the department on the ambulance stretcher at the time of admission. A little care and preparation of the patient, however, may pay dividends in the form of a more accurate appraisal. Cleansing of the colon by means of an enema should be done if not contraindicated. The urinary bladder should be emptied, by catheterization if necessary. Uniform preparation of the patient is not always practical because of the urgent nature of the case and the condition of the patient.

Positioning of the patient varies with the condition suspected, but in general a posteroanterior study of the chest with the patient standing or seated and an anteroposterior study of the abdomen with the patient both supine and erect are employed first. If the patient cannot be seated, an anteroposterior study of the chest with a long target-film distance can be substituted. Anteroposterior studies of the abdomen with the patient in both right and left lateral decubitus positions may be employed, if the erect abdominal view cannot be obtained. In the heavy patient with a broad abdomen, the flanks are better visualized when the 14 by 17 inch film is placed transversely. Diminished penetration will afford better soft tissue detail.

When free gas in the peritoneal cavity is suspected, a study with horizontal direction of the x-ray beam is essential. In this case, the abdomen can be examined with the patient erect, supine, or in lateral decubitus position. The type of examination employed may vary according to the equipment available and its flexibility. With some installations, Bucky exposures for lateral decubitus studies are not possible. A wafer grid may be used, but directing the central ray must be rather exacting or an excessive grid pattern will obscure detail in the tissues being examined.

In biliary obstruction due to a stone impacted in the gallbladder, cystic duct, or common bile duct, without peritonitis, the studies of the chest and abdomen generally show no unusual findings, except for possible opaque calculi. In suspected biliary obstruction, a left antero-oblique study of the upper abdomen is helpful in differentiating calcified costal cartilages from possible opaque calculi. The gas pattern in the gastrointestinal tract is usually not remarkable, and there are no soft tissue masses or alteration of the normal soft tissue shadows. When gaseous distention of the hepatic flexure of the colon and of the small bowel coils is present as a result of swallowed air, a pressure defect of the distended gallbladder may be brought into relief. If there is accompanying peritonitis, localized paralytic ileus may occur. In rupture

of the gallbladder, the ensuing peritonitis results in fluid accumulation in the flanks and considerable distention of the coils of the small bowel and colon.

Sudden obstruction of the ureter due to a calculus may result in symptoms resembling intestinal obstruction, with cramps, gaseous distention, and constipation. The two most dramatic cases I have encountered were caused by left ureteric calculi. The radiographs revealed excessive gas in the small bowel and colon, but the pattern was not that usually encountered in mechanical obstruction. From a clinical standpoint, considerable abdominal distention was present. The mechanism of the gastrointestinal changes is probably one of a mild degree of paralytic ileus.

The common type of intestinal perforation is that resulting from peptic ulcer. Air may or may not enter the peritoneal cavity through the perforation, depending on the content of the stomach and the position of the patient subsequent to the perforation. Examination of the chest with the patient seated or standing and of the abdomen with the patient supine is generally sufficient to demonstrate pneumoperitoneum. Anteroposterior projection with the patient in left lateral decubitus position may be more successful, especially if the patient remains in the position a few minutes before the exposure. The absence of pneumoperitoneum does not rule out the possibility of perforation. There is little evidence of gaseous distention of the intestine soon after perforation occurs, but as peritonitis develops paralytic ileus results in extensive dilatation. Other causes of pneumoperitoneum are perforations caused by trauma, carcinoma, laparotomy, and fallopian tube insufflation and perforation of Meckel's diverticulum. It should be remembered that pneumoperitoneum persists 2 weeks or more following abdominal surgery. The air in the peritoneal cavity may not be free, but air may be trapped near the point of perforation, in the lesser peritoneal sac, or beneath the liver.

Intestinal obstruction varies greatly in its roentgen appearance. The classical finding in distal small bowel obstructions has been accepted as the so-called "step-ladder" pattern of dilated gas-filled small bowel coils, with complete absence of air distal to the point of obstruction. Since most of the gas present in the gastrointestinal tract is due to swallowing of air, distention does not take place immediately after complete obstruction occurs, but it may reach alarming proportions in a relatively few hours. Simultaneously with the accumulation of gas in the intestinal tract, there is an outpouring of secretion from the glands of the gastrointestinal tract, resulting in accumulation of considerable liquid. Erect films reveal that the bowel content is more liquid than gaseous.

The location of the distended coils is no index as to the distance of the obstruction from the pylorus since developmental variations in the location of various groups of small bowel coils are frequent. A more satisfactory means of determining the level of obstruction is observation of the mucosal pattern. The mucosa in the upper small bowel is thicker than in the distal ileum, and the roentgen pattern of the valvulae conniventes is more prominent in the upper small bowel. When there is extensive small bowel distention, the location of the obstruction can be identified as an area in which there are no gas filled loops. A plausible explanation of this is that just proximal to the obstruction there is complete filling of the bowel with liquid, which remains there as a result of peristaltic activity and creates a void in the distribution of air in the lumen of the intestine. Cases of suspected intestinal obstruction should be examined in anteroposterior projection, with the patient both supine and erect. The presence of gas-fluid levels in the small bowel is practically always considered to indicate a pathologic condition.

Paralytic ileus is at times difficult to distinguish from mechanical obstruction because occurrence of the former alone is rare. In paralytic ileus both small bowel and colon distention occur as a rule, whereas in mechanical obstruction of the small bowel, distention of the colon occurs rather late. Paralytic ileus is frequently complicated by volvulus or kinking of the bowel caused by adhesive bands resulting from surgery or peritonitis. Paralytic ileus appears to occur repeatedly in patients with vitamin B complex deficiency, with no other apparent cause for the pathology. Anteroposterior studies with the patient both supine and erect are of aid in demonstrating the gaseous distention. The flanks should be examined carefully for evidence of separation of the intestinal coils, indicating fluid accumulation in the peritoneal cavity. Films in lateral recumbent position with horizontal direction of the central ray are helpful in demonstrating free peritoneal fluid. Peritoneal fluid is relatively rare in uncomplicated intestinal obstruction. Roentgen diagnosis of ascites is hazardous unless the amount of fluid is considerable.

Mesenteric thrombosis is a cause of paralytic ileus. The mechanism responsible is the disturbance of circulation in the intestinal tract resulting from the occlusion of the mesenteric vessels. Soon after vascular occlusion occurs, gas and liquid accumulate in the involved loops. The roentgen findings are variable and depend on the extent of the vascular occlusion and its location. This condition must be considered along with other causes of intestinal distention, but an accurate diagnosis can rarely be made on roentgen findings unless the cause of the mesenteric thrombosis is suspected.

Pyloric obstruction is a condition which can be detected readily from survey films made with the patient both erect and supine. Despite a history of repeated vomiting, marked gastric retention is generally present. Even when no nourishment is taken by mouth, liquid material accumulates in the stomach as a result of hypersecretion. The study of the abdomen or of the chest with the patient erect reveals a wide gas-fluid level in the upper left abdomen, signifying the dilated stomach. The amount of air in the stomach is not unusually remarkable. In the supine survey of the abdomen, the gastric contents are sufficient to cast a shadow of increased density in the upper abdomen, particularly on the left. When the pylorus is completely obstructed, there is generally little gas in the intestine.

Pancreatitis is rarely diagnosed on the basis of radiography, without contrast material. The survey film may reveal dilatation of the stomach and duodenum, as well as some gaseous distention of the remaining small intestine and colon. It is sometimes possible to delineate an enlarged pancreas between the gas filled stomach and colon. Film and fluoroscopic examinations employing a contrast meal are generally necessary for accurate diagnosis.

There are several extra-abdominal conditions which may cause acute abdominal symptoms. Among

these are compression fractures, lobar pneumonia, segmental neuralgia, and tabetic crisis. In the case of vertebral injury, particularly in the splanchnic area, the patient will often complain bitterly of abdominal distention and radiographs reveal gaseous distention of the gastrointestinal tract, similar to that observed in paralytic ileus but of a less severe degree. In segmental neuralgia and tabetic crisis, the gas pattern is not remarkable. In lobar pneumonia, so-called "peritonism" is frequent, and a survey of the abdomen reveals findings similar to those in paralytic ileus.

Rupture of the spleen, liver, kidney, urinary bladder, or any portion of the gastrointestinal tract may result from trauma. If the rupture involves a hollow viscus, the roentgen findings are similar to those encountered in perforation of a peptic ulcer, with peritonitis resulting from contamination of the peritoneal cavity. In the case of rupture of the liver or spleen, there is a loss of the sharp outline of the involved organ, displacement of adjacent structures as the extent of hemorrhage increases, and gaseous distention of the gastrointestinal tract as a consequence of peritoneal irritation, with resultant paralytic ileus. Depending on the location of the rupture of the liver or spleen, elevation of the diaphragm may occur. If rupture of the kidney is present, there is increased density in the perirenal areas, obscuring the outline of the kidney, and sometimes the psoas muscle shadow. Immediate excretory urography is indicated following survey studies if renal rupture is suspected. In the case of suspected visceral rupture, studies of the chest and abdomen are indicated, the latter with both vertical and horizontal direction of the x-ray beam, with supplementary examination in lateral decubitus position if indicated.

To obtain the most accurate information, high quality radiographs are necessary and the interpretation requires an experienced observer, who is familiar with the clinical aspects of the case. The urgent nature of the case often requires that the roentgen opinion be based on wet-film findings. For this reason, the films should be viewed under the most ideal conditions possible.

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Osseous Manifestations of Systemic Diseases in Infancy and Childhood

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This paper will be limited to a brief review of three of the more common systemic diseases of the young, with special reference to osseous changes; they are scurvy, syphilis, and rickets.

In children disturbances of the bone usually are not neoplastic and may result in diffuse skeletal involvement. They are either metabolic disturbances such as rickets or scurvy or congenital disturbances such as syphilis or some forms of bone fragility.

Scurvy.—This deficiency disease is caused by the lack of vitamin C or ascorbic acid and today is found almost exclusively in babies who have been fed formula containing pasteurized or boiled milk. The heating process may destroy enough of the vitamin C content to result in clinical scurvy. The disease may remain dormant for a period, and there are seldom symptoms, either clinical or roentgenographic, during the first 3 months of life.

The first signs are usually noted between the sixth and tenth months of life, and there may be an acute onset. Probably the earliest change is generalized bone atrophy and thickening of the epiphyseal plates (which *may* occur in other diseases also). In the roentgenogram there is a marked increase in the density of the calcification behind the epiphyseal line. This is described as the *white line.* The small epiphysis may have a characteristic ring about its edge.

Behind the dense line, areas of rarefaction are frequently seen, and at these sites epiphyseal separations may take place. When the heavy epiphyseal plates project laterally beyond the usual limits of the shaft, they form spurs and provide one of the most diagnostic roentgen features of scurvy. In the bone shaft, the spongiosa becomes atrophic resulting in the "ground glass" appearance in the roentgenogram. The cortex becomes markedly thinned and may not be visualized.

There may be subperiosteal hemorrhage along the long bones, particularly the femora, tibiae, and the humeri. Usually it is difficult to detect subperiosteal hemorrhage in the roentgenogram until healing begins and is accompanied by calcification. Fractures through the thickened and brittle epiphyseal plate are diagnostic of scurvy when syphilis can be excluded. Healing results in thickened cortex and the so-called transverse line which may be deeply buried in the shaft.

Syphilis.—In this disease the spirochetes are implanted in the metaphyses and diaphyses and produce destructive and productive changes. The outstanding characteristic is the multiple bone involvement, usually the tibiae, femora, and humeri. Roentgenographically, the syphilitic inflammation is irregularly diffuse, involving the diaphyses and metaphyses of each of the several bones affected. A curious feature of skeletal syphilis is the absence of involvement in the epiphyseal ossification centers even when the most marked productive and destructive changes are visible in the adjacent shafts. Congenital syphilis usually manifests

itself in two ways: (1) parallel periosteal reaction involving multiple long bones; (2) syphilitic epiphysitis or metaphysitis. It should be pointed out, however, that parallel type of periosteal elevation in the long bones does occur in apparently healthy premature infants. This elevation may also occur as a result of a low-grade infection in infants. Such a finding, however, should be viewed as suggestive of syphilis until proved otherwise. The lesions of the epiphyseal region are characterized by:

1. Irregular serration of the epiphyseal lines caused by saw-tooth areas of destruction in the diaphysis which usually *do not go completely* across the entire width of the bone.

2. A very small amount of parallel periosteal reaction arising over the diaphyseal region immediately above the epiphyseal line.

3. A light zone of increased calcium deposit may be present just beyond the epiphyseal line.

The shaft of the bone may be unaffected or show extensive destructive or productive changes. Scattered focal cortical destruction results in patchy moth-eaten rarefaction. There may be cortical thickening which may persist for months after the infection has subsided. Healing of the lesions is a slow process with delayed changes in the skeletal appearance. Diagnosis of syphilis in infants should be based on the evaluation of the roentgen, clinical, serologic, and bacteriologic findings.

Rickets.—The principle causes of juvenile rickets are the lack of vitamin D in the food and the lack of ultraviolet radiations of sunlight. This disease is characterized by the failure of calcification of growing cartilage and bone. Rickets is a disturbance of calcium metabolism, not of bone growth. There is a marked decalcification of the bone and cartilage which was partially calcified prior to the onset of rickets. The magnitude of the changes is directly proportional to the velocity of growth; the most marked lesions are found at the ends of the most rapidly growing bones. The roentgen findings are: a cupping and enlargement of the ends of long bones and a haziness of the diaphyseal ends of the bone. There is osteoporosis without actual bone destruction. There is bowing of the shaft of the bone with thickening of the cortex on the concave and thinning on the convex side of the bone. There usually is no expansion of the bone or invasion of the soft parts.

Differential Diagnosis.—

Rickets is characterized by:

1. Expansion of the end of the bone
2. Widened zone of epiphyseal bone growth
3. Extreme osteoporosis
4. Absence of periosteal reaction.

Scurvy is characterized by:

1. Uniform zone of destruction extending completely across the bone.
2. Appearance of double epiphyseal line.

Syphilis is characterized by:

1. Irregular, saw-toothed areas of destruction which do not extend across the diaphysis
2. Small amount of periosteal reaction
3. Light zone of calcium just beyond the epiphyseal line.

As mentioned above, we have purposely confined this discussion to three systemic diseases which show osseous changes. However, we feel that if it serves to emphasize one or more diagnostic points, its preparation has been worth while.

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Differential Diagnosis of Pulmonary Neoplasms

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Primary pulmonary neoplasms in children are rare with the exception of the lymphoma group.[1] Metastatic lesions are the most common, particularly those from Wilms' tumor, neuroblastoma, and osteogenic sarcoma. These lesions may be multiple or single and will vary in size from discrete small lesions to masses involving the entire lobe or lung. The major point differentiating a neoplasm from a pneumonic process is the absence of any associated obstructive atelectasis or emphysema. In the adult, benign tumors of the lung are very rare.[2]

I wish to discuss solitary intrapulmonary lesions, also known as "coin" lesions, which in most cases are asymptomatic. These lesions are of homogenous density, round, oval, or slightly lobulated; their edges sharply demarcated, their contours smooth, and there may or may not be calcification. These lesions present a problem in differential diagnosis. The presence of calcification cannot always be ascertained and studies other than routine roentgenograms are necessary such as the use of the Bucky diaphragm and laminagraphy, and even after these measures are performed, calcification may only be finally demonstrated in the gross pathologic specimen. Calcification within these solitary masses leads to consideration in diagnosis of hamartoma, tuberculoma, inflammatory granuloma, or metastasis from an osteogenic sarcoma. If there is no calcification, these same possibilities must be kept in mind as well as nonspecific inflammatory processes including lipoid pneumonia, abscesses, fluid-filled cystic lesions, bronchogenic cysts, bronchial adenoma, vascular lesions such as infarcts, specific infectious processes such as fungous infections, and finally bronchogenic carcinoma. Cavitary lesions within the solitary tumor indicate such possibilities as carcinoma, tuberculosis, bronchial cysts, and chronic lung abscesses. Effler, Blades, and Marks[3] reported an interesting series of asymptomatic pulmonary lesions which

were discovered by x-ray. The average age of the patients was 35.3 years. In no case was there complete agreement among the clinicians and radiologists present as to the true nature of the lesion but in every case it was considered to be benign. Although the average age of the patients was one at which malignant lesion is not likely to occur, approximately 15 per cent of the lesions proved to be cancer. Seven of twenty-four lesions were tuberculomas.

The differential diagnosis of the most common solitary pulmonary lesions and also some of the rarer ones will be considered.

Bronchial adenomas constitute approximately 10 to 12 per cent of bronchial tumors.[4] In the past these tumors were considered benign, but they are now regarded as low grade malignant lesions and are classified by some authors as grade one carcinomas[5] since they are found to metastasize to regional lymph nodes, pleurae, other bronchial regions, the liver, and vertebrae.[6] They must be differentiated from bronchogenic carcinoma and, at times, this is difficult. Bronchial adenoma is more frequent in females and occurs in younger adult life, particularly between 20 and 40 years of age.[7]

With few exceptions, the lesions are situated in the primary bronchus where they can be readily visualized with a bronchoscope. The visible tumor mass produced by an adenoma cannot be differentiated from other types of bronchial tumors by roentgenologic study alone. The final diagnosis must rest upon the bronchoscopic and histopathologic appearance. Fluoroscopy and roentgenograms taken during forced expiration aid in discovering obstructive emphysema and should, therefore, be utilized whenever partial bronchial obstruction is suspected. Bronchial adenoma should be considered as a diagnostic possibility in every young patient who presents a history of chronic cough, hemoptysis, and repeated pulmonary infection, and in the presence of atelectasis, localized wheeze, obstructive emphysema, or unilateral bronchiectasis. Surgical resection has the advantage of removing not only the entire tumor but also the infected portion of the lung and precludes the possibility of malignant degeneration, which, although relatively infrequent, cannot be entirely discounted.

Bronchogenic cyst is generally single and there are usually no symptoms until the cyst grows large enough to produce a pressure disturbance of an adjacent organ or becomes infected. The important diagnostic point is the ovoid shape of the cyst. A second diagnostic point, although less commonly seen, is the change in shape on inspiration and expiration films. The final diagnosis rests on the pathologic study of the excised cyst. Most writers agree that a bronchogenic cyst is a developmental anomaly of the embryonic tracheobronchial tree.[7] It is usually too small to cause symptoms or to be detected in the chest film during childhood and, therefore, is rarely reported in that period. When it communicates with a bronchus and is replaced by a fluid level and air, it is difficult to differentiate from tuberculosis and carcinoma.

Hamartoma of the lung is frequently composed of cartilage and primitive connective tissue, and often is found in islands of glandular tissue with fat, bone, smooth muscle, lymphoid tissue, and amorphous calcification.[8,9] It must be differentiated from bronchogenic carcinoma, solitary metastasis, bronchial adenoma, tuberculoma, and echinococcic cyst. Of aid are the facts that hamartoma is usually located peripherally or subpleurally and has a lobulated margin. Irregular patches of calcification are present in many cases and areas of lesser density near the periphery of the tumor are occasionally seen which are due to the collection of fat. The right lower lobe is the most common site. The diameter of these tumors varies from a few millimeters to a maximum of 9 cm., the majority ranging from ½ cm. to 3 cm. In the past hamartomas have been classified as chondromas because of the predominance of cartilage. Bronchial adenoma is usually more spherical, closer to the hilum, and more apt to cause obstructive change. Pulmonary tuberculoma may simulate hamartoma even to the point of calcific nodules within the mass but is usually accompanied by other signs of chronic tuberculosis. A useful point in the identification of tuberculoma is the fact that there is usually a slight degree of surrounding inflammatory reaction so that the border of the mass appears hazy. Hamartomas are essentially benign but since their potentiality for malignant changes is unknown, they are probably better removed.

Muscle tumors of the lung and other inflammatory lesions will simulate a solitary intrapulmonary lesion and the final diagnosis must rest with histologic study.

Pulmonary infarcts may resemble a mass lesion. Unless there is good reason to suspect a vascular lesion elsewhere as an exciting cause, exploration may be the only means of distinguishing an infarct from a malignant neoplastic process.[10]

Torulosis, which produces a solitary pulmonary lesion, is a rare fatal disease due to a yeastlike organism.[11] The only method of treatment is surgical resection.

Tuberculoma[12] is usually a clinically quiescent tuberculous focus and one of the most frequently seen of the solitary pulmonary lesions. The basic pathologic process is apparently a localized area of caseous tuberculous pneumonia which may be seen in all stages of healing or progression. A review of the roentgenograms of patients in a series studied by Isaac and Ottoman[12] even in retrospect emphasizes the difficulty of differentiating between tuberculoma and a peripheral neoplasm. In addition, the presence of calcium in the lesion, a finding often emphasized as characteristic of tuberculoma, was of little help. One of the most difficult aspects of the problem of identifying a solitary tuberculoma radiologically is the frequent absence of any correlative evidence of tuberculosis. Even in the presence of suggested apical infiltrates, however, it is extremely difficult, if not impossible, to make a definitive diagnosis of such solitary radiologic shadows without exploratory thoracotomy. If tuberculomas were universally quiescent and completely hyalinized or calcified, there should be little reason to remove them. It was demonstrated that all the cases in Isaac and Ottoman's series contained caseous areas. The danger of recrudescence or dissemination was apparent. Tuberculoma appears to be a postprimary lesion arising either from exogenous reinfection or focal extension from a primary focus, probably the former.

The differential diagnosis of a pulmonary neoplasm in the cavitary form is difficult. There is no roentgenologic method by which early bronchogenic carcinoma can be reliably differentiated from pulmonary infection. Bronchoscopy with biopsy is still

the only means, except exploratory thoracotomy, by which a positive diagnosis can be made or metastatic lesions can be discovered.[13] Differentiation between an infection and a malignant neoplasm may require serial roentgenograms which may mean weeks, and even months, of delay, although the finding of tumor cells in the sputum by special staining methods holds great promise as an early diagnostic aid. The type of malignant pulmonary lesion most often confused with infection is the cavitary form of bronchogenic carcinoma. The cavity is often eccentric and the contours of the inner wall have an irregular, ragged, bumpy appearance corresponding to their regular tissue necrosis within the tumor. This is especially well brought out by body section roentgenography. The irregular bumpy character of the cavity wall in neoplastic disease is not claimed to be pathognomonic of cancer for it may at times be found in other conditions, especially tuberculosis.

Cough, hemoptysis, pain in the chest, and weight loss in a patient over 40 years of age must be assumed to indicate a pulmonary neoplasm until proved otherwise. Other causes for cavitation are aspiration pneumonitis, septic emboli, nonseptic emboli which became infected, mycotic disease, foreign bodies in the bronchi or lung parenchyma, benign and malignant neoplasms. The cavities of coccidioidomycosis are neither infrequent nor difficult of recognition. They are characteristically thin walled, resembling cysts or blebs.[14] The most important thing in viewing a parenchymal cavity is always to keep neoplasia in mind. Particular attention should be directed toward the bony thorax since evidence of metastasis will save needless surgical intervention or limit intervention to palliative drainage. Observation of a known intrapulmonary lesion of unknown character should never last for more than 1 month. If it persists for that period complete investigation, including thoracotomy, should be carried out if necessary to establish an exact diagnosis.[15] Virus pneumonia, atypical pneumonia, and patchy pneumonitis run their natural courses within 3 to 4 weeks. The difference between primary and secondary pulmonary infection is that in the former the pulmonary density nearly always disappears entirely within 4 weeks, but in the latter residual density persists.

The hilar mass presents a problem in differential diagnosis.[16] The typical appearance of bronchogenic carcinoma of the hilar type in the beginning stage consists of a large, irregular density in the hilum, spreading out fanwise with strands radiating outward to the periphery of the lung fields. This shadow represents both the tumor growth in the bronchus and the accompanying atelectasis and, possibly, lymph node involvement. The extension of the neoplasm by lymphatic spread, in addition to the accompanying inflammatory change, also contributes to the composite density in the hilar region. Pulmonary tuberculosis in this region may also give the characteristic findings of bronchogenic carcinoma. Atypical pneumonia can also have the same shadow. The conditions that might cause confusion in the diagnosis are central pneumonia, Löffler's eosinophilic syndrome, primary hilar tuberculosis, lung abscess, and delayed resolution of pneumonia, all simulating bronchogenic carcinoma.

Pulmonary adenomatosis[17-19] continues to cause disagreement as to its place in the classification of neoplasms, the correct nomenclature, the origin of the basic cell, and even the existence of the condition as a separate entity. Some authors believe the tumor must be considered precancerous, that pulmonary adenomatosis and alveolar cell carcinoma are not the same disease but are closely related. Pulmonary adenomatosis occurs with about equal frequency in males and females. The majority of cases have occurred between the ages of 40 and 70 years. At present there seems to be no method of treatment which will halt the spread of the disease. Dyspnea is its most constant feature.[19] This can be explained by the fact that the hyperplasia of the epithelium in the alveoli interferes to a great extent with the gaseous exchange between the alveoli and the pulmonary capillaries. Furthermore, the accumulation of mucoid material in the bronchi interferes with the passage of air to the alveoli. Cough is also a constant and striking feature. The clinical findings are of little help in the diagnosis of benign pulmonary adenomatosis. The temperature is normal unless complications such as pneumonia are present. The laboratory studies are all within normal limits. The duration of symptoms is from a few months to 2 or 3 years, the average being about 1 year. The patient dies as a result of asphyxiation and terminal pneumonia.

X-ray findings show that pulmonary adenomatosis is extensive and involves considerable portions of both lungs. The areas of greatest involvement are in the central lung zones and in the bases. The roentgen pattern is of two types. In the first type, the areas involved show innumerable nodular densities. These nodules cast a relatively soft shadow and are rather poorly delineated, fading somewhat indistinctly into the normal lung tissue. They vary in size from about 2 mm. to about 1 cm. in diameter. The other type pattern is a homogenous density resembling areas of pneumonic consolidation; the lesions progress rather slowly, and serial films over a period of several weeks show very little change. A negative finding of considerable importance is the absence of hilar or mediastinal adenopathy. The absence of pleural effusion is also worthy of note. Although a single roentgenogram of the chest is not characteristic of pulmonary adenomatosis and is therefore not diagnostic, a correlation of the clinical features with the roentgen findings of serial examinations should lead to the diagnosis in a fairly high percentage of cases.

Pulmonary adenomatosis is a rather rare disease of unknown etiology characterized by a remarkable proliferation of alveolar lining cells throughout large areas of the lungs, by an absence of invasive characteristics, and by an absence of regional or distant metastasis. The most striking finding in this examination is the tremendous quantity of mucoid sputum aspirated from the bronchi.

Apical lung cancer,[20] occurring in the region between the root of the neck and the apex of the lung, is characterized by a peculiar clinical syndrome resulting from involvement of the brachial plexus and of the cervical sympathetic chain. This consists of pain around the shoulder and down the outer side of the forearm of the affected upper extremity, atrophy of the muscles of the hand, and Horner's syndrome. Pancoast designated these tumors as superior pulmonary sulcus tumors.[2] The *x-ray findings* denote sharply defined shadows at the apex of the lung with destruction of one or more of the upper three ribs posteriorly and some erosion of the transverse processes and bodies of the corresponding vertebrae. There

is slight indentation and displacement of the trachea to the opposite side. Metastatic lesions from the breast, kidney, adrenal, pancreas, and stomach, thymic tumors, and intrathoracic sympathicoblastomas have been reported as causing this syndrome. The first symptom in practically all cases is shoulder girdle pain which, sooner or later, radiates down the arm. Patients having shoulder girdle pain should have an x-ray examination instead of being treated for rheumatism, arthritis, neuritis, bursitis, and the like for months before adequate examinations are made.

The instillation of iodized oil into the bronchi is a feasible and highly accurate procedure.[9] With the exception of bronchoscopy it is, no doubt, the most useful method for the delineation of an intrabronchial tumor. There are certain contraindications to bronchography, and furthermore, the procedure is not without complications and difficulties so that it is not universally applicable. If dyspnea is present, there should be hesitation about administering iodized oil. Fever and toxicity are important contraindications. Many thoracic surgeons object to bronchography in carcinoma of the lung because they feel surgery should be delayed for 6 weeks or more after the use of the oil. Such a period of waiting allows further increase in the size of the tumor. For delineation of the bronchi, without the use of oil, body section x-ray has been found to be most useful.[21] It is not intended to replace older methods of diagnosis, but it is extremely useful and in certain cases more accurate than older methods and represents an adequate supplementary procedure.

A chest film should become as much a part of the laboratory examination of every patient admitted to a hospital, clinic, and doctor's office as the urinalysis, blood count, and blood test. From 8 to 22 per cent of all general hospital patients show chest disease on routine admission films. This percentage of positive findings is higher than with any other routine accepted procedure.

I must emphasize an oft repeated axiom that a shadow of any character seen in the roentgenogram is merely a record of gross pathology as reflected in the shadow contours, position, and density. Knowing that carcinoma can at some time or other mimic almost every other chest condition, the roentgenologist must use caution before making a dogmatic diagnosis. The roentgenogram, however, still remains the only means of making the early diagnosis of a lesion which is peripheral and beyond the reach of the bronchoscopic observer and beyond the scope of other laboratory examinations.[22] Neither bronchoscopic examinations nor the study of bronchial secretions and sputum tells the entire story, even though each has served to increase greatly the accuracy of diagnosis of chest conditions. However, the point has not yet been reached when every atypical lesion which the bronchoscopist or the pathologist studies in this manner can be diagnosed unequivocally. Thus the surgeon will have to do an exploratory operation in those cases in which roentgenograms show findings highly suggestive of neoplasm and in which study of the bronchial secretions, sputum, or the bronchoscopic examination is indeterminate. The more chest lesions that are seen and the more thoroughly each case is studied, the greater will be the number of cases in which confusion may exist. Once the question of a neoplasm has been raised, the physician must not temporize.

It has been repeatedly shown that the lesions which roentgenologically most readily simulate cancer of the lung are usually best managed surgically. A small mass seen on the x-ray which fails to grow appreciably in a few months or even a year or more does not exclude carcinoma of the lung. A small untreated cancer of the lung may develop metastases while remaining almost stationary in size. Failure to appreciate these facts can result in long delay in some of the most favorable cases from the standpoint of pathology. A sharp border to a rounded mass should not be considered an indication that the lesion is benign. A negative bronchoscopic or cytologic report may be of limited significance in ruling out early cancer of the lung. It is believed that watchful waiting in such cases may be a highly dangerous procedure since stability on x-ray examination even for a prolonged period does not disprove cancer, and roentgenologic evidence of progression may already indicate inoperability. At present, early exploration of the chest offers the only possibility of early diagnosis and early treatment in the majority of cases.[22]

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Radiographic Diagnosis of Urinary Tract Neoplasms in Infants and Children

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Neoplasms of the urinary tract in infants and children and the lesions from which they must be differentiated present a triply faceted problem. First, the average radiologist encounters too few of these tumors to become thoroughly familiar with their diagnostic features, so that he is at somewhat of a disadvantage in such a situation. Second, if he is cognizant of the histopathology, natural history, and biology of this group of entities, he is distinctly aware that he may be dealing with the most malignant lesions known to medical science. Third, the fact that an entire lifetime may be salvaged if the lesion is quickly and properly identified is sufficiently stark in its implications to elevate this subject to a place of importance not indicated by its relatively uncommon occurrence. I do not propose to discuss treatment, but a report in a recent journal[1] of a marked improvement of survival figures in recent years should be an impetus to review the subject and to contribute to a more optimistic prognosis by more competent diagnosis.

Tumors of the urinary tract in children, other than Wilms' tumors, are curiosities. Sarcomas of the urinary bladder and prostate are extremely rare. Ureteric tumors are even more unique. However, extraurinary tract lesions in the upper abdomen, both intraperitoneal and retroperitoneal, are common, and their differential diagnosis from Wilms' tumor is a relatively frequent problem.

Thus, the most important consideration in this field is Wilms' tumor and the lesions with which it may be radiographically and clinically confused. Those occurring with relative frequency in pediatric practice are a tumor, adrenal or retroperitoneal neuroblastoma, and a non-neoplastic kidney enlargement, hydronephrosis, usually congenital. Those of lesser incidence include benign renal tumors, congenital polycystic disease, retroperitoneal masses outside the kidney and adrenal gland such as lymphoblastomas, lipomas, myomas, and cysts, neoplastic or inflammatory hepatic enlargements, splenomegaly, pancreatic cysts and tumors, ovarian tumors, and fecal and intestinal tumors.

Wilms' tumor presents radiographically as an enlarging, distorting kidney lesion in an infant or child who has a palpable abdominal mass which frequently has been discovered accidentally during routine handling in the course of daily living.

The kidney shadow on the involved side may be altered in contour throughout or only at one pole or at its midportion. It may be only slightly larger than average or it may be massive enough to produce a density filling half the abdomen, displacing the gas-filled intestine downward and to the opposite side, elevating a diaphragmatic leaflet, bulging the flank shadows, and compressing the liver or spleen upward and anteriorly. Often a definite renal shadow is not seen, and a large, ill-defined density is present on the involved side.

The renal shadows in infants and children are normally relatively large and the lower poles low lying, being below the iliac crests in some infants. Under 5 or 6 years of age, the renal shadows are somewhat irregular because of the persistence of lobulations. The perirenal fat is scant until the eighth year, resulting in relatively poor visualization of the kidney outlines. If the entire normal renal outline can be visualized through the abnormal density, the probability of Wilms' tumor diminishes. Calcification may be present in a kidney neoplasm, although its frequency is probably greater in adrenal tumors. A large liver shadow will produce a density similar to that of a large, right-sided Wilms' tumor, especially in younger children whose paucity of perirenal fat makes the contrast between normal kidney and large liver poor. The abnormal shadow resulting from a neoplastic kidney is unilateral, although it may cross the midline. Bilateral kidney enlargement is almost never the result of Wilms' tumor but usually represents congenital cystic disease. Splenomegaly, such as occurs in anemias or lymphoblastoma, may produce an ill-defined left abdominal density overlying the renal area and cause confusion, but often the spleen may be identified by its characteristic contour and disturbance of the gastrointestinal air pattern.

The presence of some or all of the features mentioned, when seen on survey films of a young patient, should suggest the possibility of kidney tumor, and further urinary tract study should be undertaken.

The urographic manifestations of Wilms' tumors are protean. They range from minimal, minor, calyceal flattening or compression to bizarre patterns of grossly distorted and displaced calyces, pelvis, and ureter, and even to absence of dye if obstruction of the pelvis or ureter by the encroaching mass has occurred. Closer analysis of the patterns which may present is described by Campbell.[2] He describes elongation of calyces, pelvic filling defects, enlargement of the pelvis, compression and flattening of the pelvis, and ragged pelvic borders due to ulceration and infection. The calyceal elongation is supposedly due to tumor growth outward toward the periphery of the kidney, with the ultimate production of a narrow streak of dye and fading out of the terminal calyces. Urograms are important in demonstrating the opposite kidney since its presence and normalcy have a bearing upon management.

The only other enlarging renal lesion which occurs frequently in children is hydronephrosis, usually the result of a congenital ureteropelvic or ureteric obstructive mechanism. A large percentage of such cases results in nonvisualization of the affected kidney by the intravenous method. Hydronephrosis is the leading single cause of nonvisualization of kidney in infants and children. Consequently, nonvisualization, in the presence of a large kidney shadow, suggests a congenital obstruction rather than tumor. When dye is seen in the collecting system, it appears as the

familiar, rounded, ill-defined shadow. Retrograde study usually will confirm and delineate the obstruction; it may visualize the dilated collecting system or demonstrate the deformity due to tumor more sharply.

Further essential roentgenographic evidence in suspected renal tumors is obtainable by surveying the lungs and skeletal system. Typical metastatic patterns in Wilms' tumor are the presence of lesions in the liver and in the pulmonary fields where they are seen as discrete, rounded masses, often in an apparently hematogenous distribution throughout both fields. Solitary lesions or a few masses may also be seen. The skeletal system is often free from evidence of secondary malignancy until late in the course of the disease.

The demonstration of some or all of these findings in an infant or child, usually not more than 8 years of age, presenting either an asymptomatic, large, easily palpable abdominal mass which gives a pot-bellied appearance or a similar mass associated with vague gastrointestinal or urinary complaints, occasionally with weight loss and hematuria, suggests a Wilms's tumor.

Most important of the extrarenal lesions to be considered in differential diagnosis is the adrenal neuroblastoma, which is about equal in incidence to Wilms' tumor in infants and children. Together these tumors comprise a large percentage of malignancies encountered in pediatric practice. The child with adrenal neuroblastoma usually is pale, apathetic, and wasting, as compared to the Wilms' tumor victim who usually appears to be in relatively good health. A mass is palpable in the abdomen but is sometimes more lateral and posterior in location than a renal mass. Some children complain primarily of back pain, leg pain, and weakness. Ocular signs such as discoloration of periorbital tissues and unilateral exophthalmos are suggestive of adrenal lesions.

Abdominal survey films show a density in the upper abdomen which is often indistinguishable from that produced by a Wilms' tumor. Again, if the kidney shadow shows through the density or appears to be depressed by it, an extrarenal lesion, presumably adrenal, can be considered. The incidence of intratumoral hemorrhage is greater in adrenal than renal lesions so that calcifications are more often seen in suprarenal tumors. However, the shadows produced are essentially the same.

Urographic findings may also be analogous in spite of the difference in origin of the tumors. Occasionally an adrenal mass will displace the kidney downward and the liver or spleen upward, while flattening the most superior calyx and leaving the remaining calyces unaltered. This is fairly typical, yet even with this picture the exact identification of the disease is difficult.

The extra-abdominal roentgen findings are sufficiently important to be searched for diligently and in many cases may supply the extra bit of information necessary for proper diagnosis. Their significance lies in the fact that the average metastatic pattern of Wilms' tumor differs from that of the adrenal lesion. The kidney neoplasm usually metastasizes to liver and lung and rarely to the skeletal system until very late. When bone lesions do occur, they are usually in the pelvis. Neuroblastomas, on the other hand, metastasize to bone early—so early in some cases that the presenting complaint is of a skeletal disturbance and the initial findings are osseous destructive lesions suggestive of secondary malignancy. Most frequently the long bones and skull are involved. Sometimes the long bone lesions are bilaterally symmetrical and may simulate those of leukemia to an extraordinary degree.

Urinary bladder tumors are rare, and usually are sarcomas which arise from the bladder wall primarily or are prostatic in origin and involve the bladder by direct extension. They are most frequent under the age of 2 and are highly malignant, progressing rapidly by direct extension into perivesical tissues. Soft, friable, and highly vascular, they herald their presence by persistent urgency, frequency, and dysuria, incontinence, and recurrent hemorrhage. Pyuria is quick to develop, and often a suprapubic mass is palpable soon after symptoms are recognized. Urography and cystography demonstrate filling defects, usually basal in location, and local or generalized mural irregularity. A small bladder with evidence of a mass often signifies complete infiltration of the wall. Ureterectasis, pyelectasis, pyelonephritis, and poor kidney function may be associated findings.

I will now present a few cases which demonstrate some of the features just discussed.

Case 1. A Wilms' tumor was discovered in a 3-year-old girl whose father, a physician, felt a left-sided abdominal mass while bathing the child. The girl had evidenced no symptoms. Roentgen studies were undertaken immediately. The survey film demonstrated a large, soft tissue mass in the left abdomen, its lower margin extending down to the iliac crest. The left psoas shadow was obliterated, the left flank bulged outward, and intestinal gas shadows were displaced downward and to the right. Urography showed gross distortion, compression, and caudal displacement of the dye shadows in the left kidney. The right kidney was normal. In the lateral projection, the large dense mass lay more posteriorly than anteriorly, and the distorted dye shadows were at its lower pole. The lumbar spine appeared to be free of metastasis. A chest film was negative. The left kidney was resected immediately, and a Wilms' tumor found. Postoperative x-ray therapy was administered, but 6 weeks later, a chest film showed multiple nodular densities suggestive of hematogenous metastasis. The patient expired shortly afterwards.

Case 2. A Wilms' tumor was found in a boy of 12, which is somewhat older than the average age of such patients. A lateral survey film with a catheter in the left ureter showed a large, dense, posteriorly located upper abdominal mass containing two calcifications. The catheter was displaced posteriorly in its upper half. The calyces were incompletely visualized, and those seen were elongated and compressed. The lateral view showed the posterior displacement of the upper half of the collecting system. The right kidney was normal. An apparent pressure defect was seen upon the greater curvature of the air-filled stomach. Subsequent autopsy proved this to be a Wilms' tumor with contained calcifications.

Case 3. Admission films of the abdomen and chest of a 3-year-old boy showed a large right upper quadrant soft tissue density, bulging of the right flank, obliteration of the right psoas shadow, and displacement of the intestinal coils. The right diaphragm appeared to be elevated. A vague density was present in the lower right lung field and a definite nodular shadow in the left apex. This combination of upper abdominal mass and nodular pulmonary lesions in a

young patient was considered to be caused by a Wilms' tumor. On this basis, radiation therapy was instituted, and 2 months later, the lung fields were negative. However, the patient died, and autopsy confirmed the clinical impression of Wilms' tumor.

Case 4. A 3-year-old girl with an asymptomatic palpable Wilms' tumor presented many criteria of significance. The initial procedure was a colon study. There was an extrinsic pressure defect on the superior margin of the left transverse colon and the proximal sigmoid was displaced upward and toward the midline. The entire upper colon was displaced anteriorly. Thus there seemed to be a large, posteriorly located, left-sided mass lying extrinsic to and behind the colon.

A urogram produced the following findings: On the survey film there was a homogeneous soft tissue density in the left upper and midabdomen. The intestinal air pattern was unremarkable. The lower pole of the mass was at a slight distance below the iliac crest. Its lateral extent obliterated the properitoneal fat line and bulged the left flank. At its medial margin a thin linear calcification was present, overlying the second and third lumbar bodies. The left psoas margin was less distinct than the right. The 5-minute film showed a bizarre dye pattern in the upper third of the mass and demonstrated medial displacement of the left ureter, especially in its lower portion. The 15-minute study again showed gross pelvic and calyceal distortion with some suggestion of elongation and thinning of the major calyces and compression of the inferior minor calyces. The right kidney appeared normal. Lateral projection located the mass posteriorly, displacing the air-filled intestine anteriorly, and showed the markedly elongated, posteriorly displaced calyces. A survey film made after 5,100 r in air had been delivered to the lesion showed its dramatic regression in size and demonstrated with great clarity the calcification of its medial border.

Case 5. A 7-month-old female with an asymptomatic, palpable, abdominal mass was seen. A survey film with a catheter in the right ureter showed a huge, rounded, right abdominal density extending across the midline and into the pelvis, displacing the gas-filled bowel downward and to the left and the ureter to the opposite side. The filled study demonstrated cephalic displacement of the calyceal system and obliteration of the inferior calyces. These films seemed to substantiate the clinical opinion that a Wilms' tumor was present. Surgical exploration uncovered a large renal cyst which was drained and resected. Although many of the criteria for a Wilms' tumor were found present, a rounded lesion with such homogenous density probably should have suggested a cystic mass. However, an occasional Wilms' tumor may become cystic.

Case 6. A large palpable intra-abdominal mass in a 4-year-old girl was difficult to identify before surgical exploration. Pyelography suggested medial displacement of the upper third of the left ureter and alteration or poor filling of the right minor calyces. Urography was also only suggestive of poor filling of the right kidney as compared to the left. A colon examination showed an extrinsic pressure defect on the superior border of the hepatic flexure. Reexamination by the retrograde method showed a dense right upper quadrant without intestinal air shadows and obliteration of the right psoas shadow. The left kidney shadow was essentially normal—the right was not visualized. The filled study again suggested blunting of the terminal calyces and lateral displacement of the middle third of the right ureter. Thus far, the accumulated roentgenographic evidence was inconclusive, being compatible with a right abdominal mass of hepatic, renal, adrenal, or other origin. Transperitoneal operation revealed a retroperitoneal mass which was radiated and then resected by a retroperitoneal approach. The pathologist recorded the lesion as a sympathicoblastoma of retroperitoneal, probably adrenal, origin, which had secondarily invaded the right kidney.

Case 7. A young boy was brought to the hospital with an essentially asymptomatic large, palpable mass in the abdomen. The initial survey film demonstrated a homogeneous, dense soft tissue mass in the right upper and midabdomen, with displacement of intestinal loops downward and to the left. The kidney shadow was not visualized through the density. Provisional diagnoses from clinical and roentgenologic findings listed renal tumor as the leading probability, followed by a retroperitoneal mass of some other nature.

A colon study showed failure of the cecum to fill, resulting in a defect which the fluoroscopist regarded as probably extrinsic. A retrograde pyelogram showed apparently normal filling of the right kidney and ureter. In view of the pyelogram, a Wilms' tumor was felt to be improbable, and some other mass lesion involving the cecum was considered. Chest films on two occasions suggested mediastinal widening and left diaphragmatic elevation. Biopsy of a cervical node and of a retrocecal mass proved the lesion to be a lymphosarcoma.

Case 8. A severely ill infant was seen with a large, anteriorly bulging abdominal mass. A colon study showed the massive upper abdominal density bulging both flanks and depressing the hepatic and splenic flexures. No definite renal shadows could be discerned through the density. Since Wilms' tumor is rarely bilateral, it was thought that the density might represent liver, although a kidney tumor with metastasis to the upper abdomen could not be ruled out. Urography demonstrated normal renal structures and a sufficiently good nephrogram was obtained so that an essentially normal kidney shadow could be identified through the mass. Wilms' tumor was ruled out since, even if the pelvis and calyces are normal, the renal outline is altered in its presence. Abdominal exploration revealed a massive primary liver tumor.

Case 9. Wilms' tumors do not, as a rule, regress in size or disappear spontaneously. A boy presented a palpable large left abdominal mass which disappeared on several occasions only to recur. This was more suggestive of a hydronephrotic lesion than a Wilms' tumor.

Urographically, at 10 minutes, the left kidney was not visualized. This also is characteristic of hydronephrosis, which is the leading cause of nonvisualization in children. At 45 minutes, a large collection of low density dye had begun to appear in the left kidney area, and the left ureter appeared to be displaced laterally above the iliac crest. At this point, the impression from the roentgenologic evidence was unilateral polycystic kidney, with the reservation that a retroperitoneal mass involving the kidney secondarily or a primary kidney tumor could not be ruled out. Retrograde study showed an amorphous

collection of dye in the left kidney area. At surgery, a congenital band at the ureteropelvic junction, with hydronephrosis, was found.

Case 10. An 8-year-old girl was hospitalized because of suspected acute appendicitis or intestinal obstruction. She apparently presented no genitourinary symptoms. Abdominal survey film showed a large left kidney shadow having the upper pole at the eleventh to twelfth dorsal level and the lower pole at the fourth to fifth lumbar level. Its contour was average, a factor against a diagnosis of neoplasm. However, a large asymptomatic kidney in a child this age should always alert the examiner to the possibility of tumor. Urography failed to visualize the left kidney, suggesting hydronephrosis as the most likely possibility but not eliminating tumor. Retrograde study showed gross dilatation of the left pelvis and calyces and tortuosity of the proximal ureter. A hydronephrotic lesion was now established, and a congenital anomaly, at or near the hilum, was suspected. Urography was repeated, and a 2-hour film showed the hydronephrotic sac without visualization of the ureter. It was felt that the presence of a congenital ureteropelvic obstruction had been established, and exploration demonstrated an aberrant vessel.

This case is a fine example of a large kidney of normal outline, nonvisualized at initial urography, which presented almost textbook characteristics of congenital obstruction rather than of Wilms' tumor.

Case 11. Congenital deformities such as ureteropelvic obstruction are relatively common. Other more unusual defects often provide pediatric practice with a fascination not encountered in adult problems and also provide apparent evidence on which to base a false diagnosis of renal or adrenal tumor. As an example, a 5-day-old boy was practically anuric after having voided blood-tinged urine on the first day of life. A large abdominal mass was palpable. The survey film was interpreted as demonstrating a left abdominal mass and intestinal air displacement to the right. The impression from clinical and roentgenologic findings was probable left renal neoplasm. The infant expired before urography could be performed, and autopsy revealed a congenital atresia of the posterior urethra, bilateral hydronephrosis, and enormous dilatation of the left ureter, which was the size of a lemon. Both kidneys were large and cystic. Had urography been possible, the probable demonstration of either bilateral hydronephrosis or nonfunctioning kidneys would have been helpful in ruling out neoplasm.

Case 12. Anomalies outside the urinary tract may also be involved in differential diagnostic problems of kidney tumors, as in the case of a male newborn who could not retain feedings, who was recurrently cyanotic and presented a large palpable left abdominal mass which increased in size over several days. Abdominal survey films showed a left upper midabdominal density with a well-defined lower border and displacement of the air-filled stomach upward and to the right and of intestinal coils downward. The psoas shadows were indistinct and no definite renal outlines were visible. Lateral projection seemed to place the mass behind the stomach in the upper abdomen. A provisional diagnosis of Wilms' tumor was entertained. Urography was attempted but was unsuccessful. At a large children's hospital, to which the patient was transferred, preparation was made for a renal exploration. On the morning of surgery the infant vomited fresh flood. The surgeon then elected to use an anterior approach, and found a reduplication of the stomach which contained a large amount of retained secretion, accounting for the clincally recognized rapid increase in size of the mass. Clinically a rapid progressive increase in size of Wilms' tumor is unusual although it may occur as a result of intratumoral hemorrhage. If palpation and observation give the impression of a secreting tumor, other lesions should be considered.

Case 13. In a 1-year-old girl who had experienced sudden, rather massive hematuria, a urogram demonstrated an extensive, irregular filling defect at the base of the bladder. The oblique view suggested infiltration of almost the entire vesical wall. The multiple rounded defects in the dye pattern seemed to indicate the presence of a possible polypoid neoplasm. Malignant cells were found in the urine and were thought to be sarcomatous. No biopsy was obtainable as permission for cystoscopy was refused, but the defects were typical of sarcoma, and the cytopathology seemed to confirm the impression.

SUMMARY AND CONCLUSION

An attempt has been made to review the salient features of an important pediatric problem. Frequently in pediatric practice it is found that young children with renal disease cannot describe their pain with any greater accuracy than to say "I have a stomach ache." The inability of these patients to lead the way must be compensated for by rendering to them the ultimate in diagnostic acumen, since their hope for the future rests with the examination of today.

59 E. 54th St.

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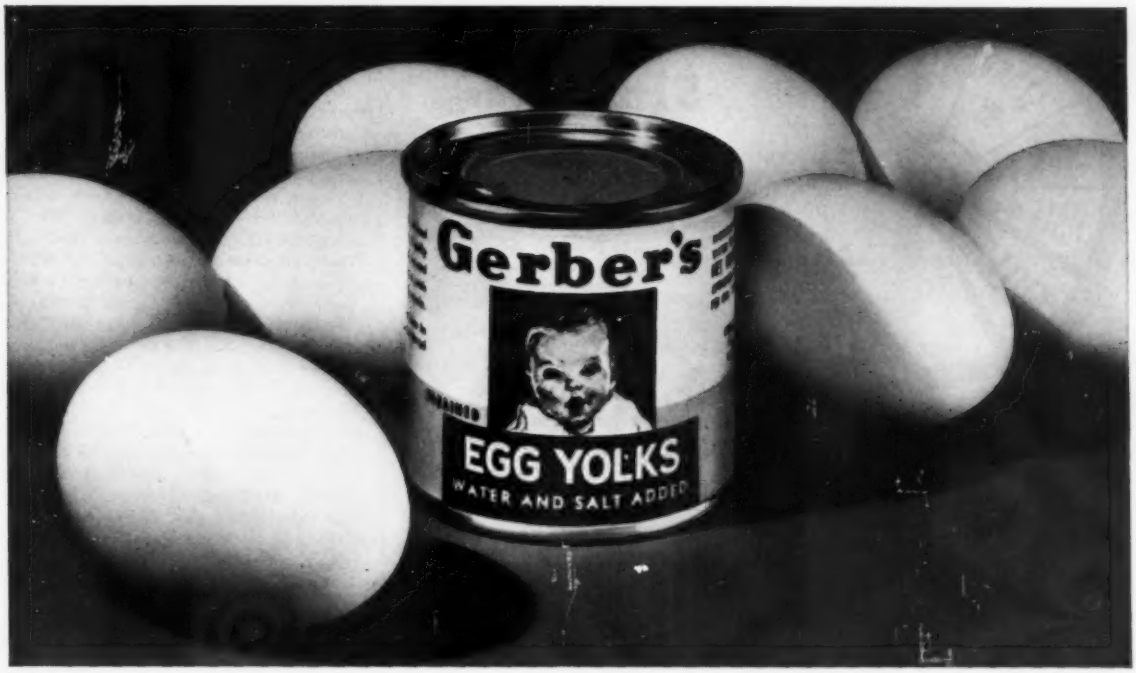

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Conventions and Meetings

Announcements

American Osteopathic Association, Fifty-Eighth Annual Convention, Royal York and King Edward Hotels, Toronto, Canada, July 12-16, inclusive. Program Chairman, Campbell A. Ward, 51 Cass Ave., Mt. Clemens, Mich.

American College of Osteopathic Obstetricians and Gynecologists, annual meeting, Hotel Statler, Los Angeles, February 16-19. Secretary, Arthur A. Speir, Box 57, Merrill, Mich.

American College of Osteopathic Surgeons, annual meeting, Hotel Statler, Los Angeles, October 18-22. Secretary, Orel F. Martin, Box 474, Coral Gables 34, Fla.

American Osteopathic Academy of Orthopedics, annual meeting, Hotel Statler, Los Angeles, October 18-22. Secretary, J. Paul Leonard, 2673 W. Grand Blvd., Detroit 8, Mich. Associate Program Chairman, Walter R. Garard, 809 S. Hobart Blvd., Los Angeles.

American Osteopathic College of Radiology, annual meeting, Hotel Statler, Los Angeles, October 18-22. Secretary, H. Miles Snyder, 5435 Woodward Ave., Detroit 2, Mich.

American Osteopathic Hospital Association, annual meeting, Hotel Statler, Los Angeles, October 18-22. Executive Secretary, Mr. R. P. Chapman, 1011 Kahl Bldg., 326 W. Third St., Davenport, Iowa.

American Osteopathic Society of Anesthesiologists, annual meeting, Hotel Statler, Los Angeles, October 18-22. Secretary, Crawford Esterline, Box 155, Kirksville, Mo. Program Chairman, Amanda C. Marshall, 766 S. Kingsley Dr., Los Angeles 5, Calif.

American Osteopathic Society of Proctology, annual meeting, Hotel Jefferson, St. Louis, Mo., April 29-May 1. Secretary, Carl S. Stillman, Jr., 3523 Fifth Ave., San Diego 3, Calif. Program Chairman, Felix D. Swope, 609 Farragut Medical Bldg., Washington 6, D. C.

Canada, annual meeting, Chateau Laurier, Ottawa, October 15-17. Secretary, Miss Joyce Currie, 609 Medical Arts Bldg., Montreal. Program Chairman, Douglas F. Lauder, 343 Queens Ave., London, Ontario. Exhibits Chairman, D. E. Firth, 80 King St., Toronto.

Georgia, annual meeting, Radium Springs, May 7-8. Secretary, Kenneth H. Wiley, 806 Mortgage Guarantee Bldg., Atlanta 3.

Hawaii, annual meeting, Waikiki, Oct. 26-28. Secretary, J. W. Stella, Room 38, Young Hotel Bldg., Honolulu 9. Program Chairman, C. W. Wyman, 417 National Bldg., Honolulu 13.

Indiana, annual meeting, French Lick Springs Hotel, French Lick, April 25-27. Secretary, F. A. Turfler, Jr., 107 S. William St., South Bend.

Kentucky, annual meeting, Brown Hotel, Louisville, Sept. 16-17. Secretary, Harold D. Benteen, 2048 Winchester Ave., Ashland. Program Chairman, Martha Garnett, 417 Fincastle Bldg., Louisville.

Maine, midyear meeting, Elmwood Hotel, Waterville, December 3-5. Program Chairman, Edward G. Drew, 166 Silver St., Waterville. Annual meeting, Hotel Samoset, Rockland, June 17-19. Executive Secretary, Roswell P. Bates, 72 Main St., Orono.

Michigan, annual meeting, Civic Auditorium, Grand Rapids, Sept. 28-30. Executive Secretary, Mr. Harve L. Smith, 13535 Woodward, Highland Park. Program Chairman, L. W. Pettycrew, 1205 N. Michigan Ave., Saginaw. Exhibits Chairman, M. B. Goldberger, 2730 S. Washington, Saginaw.

New Jersey, midyear meeting, Ritz Carlton Hotel, Atlantic City, September 12-13. Executive Secretary, Mr. I. J. Tecker, 21 Hancock St., Riverside.

New York, annual meeting, Hotel Statler, New York City, October 15-17. Secretary, Robert E. Cole, 417 Main St., Geneva. Program Chairman, W. Kenneth Riland, 71 Broadway, New York 6.

North Carolina, annual meeting, Battery Park Hotel, Asheville, October 16-17. Secretary, S. D. Foster, 710 Public Service Bldg., Asheville. Program

Chairman, Elizabeth Smith, 414 Wachovia Bank Bldg., Asheville.

Ohio, refresher meeting, Hotel Carter, Cleveland, October 6-7, annual meeting, Columbus, May 2-5. Executive Secretary, Mr. William S. Konold, 50 E. Broad St., Columbus. Program Chairman (annual meeting), Donald J. Ulrich, 141 E. Main St., Kent.

Oklahoma, annual meeting, Tulsa, November 3-5. Executive Secretary, Mr. Walter E. Gray, 210 Braniff Bldg., Oklahoma City. Program Chairman, E. C. Baird, 202 Thompson Bldg., Tulsa 3.

Osteopathic College of Ophthalmology and Otorhinolaryngology, annual meeting, Hotel Statler, Los Angeles, October 22-24. Executive Secretary, Mr. William S. Konold, 50 E. Broad St., Columbus 15, Ohio. Program Chairman, J. E. Leuzinger, 1813 Pine St., Philadelphia 3.

Rocky Mountain Osteopathic Conference, Broadmoor Hotel, Colorado Springs, November 13-15. Secretary, C. Robert Starks, 1459 Ogden St., Denver 18.

Vermont, annual meeting, Montpelier Tavern, Montpelier, September 30-October 1. Secretary, Howard I. Slocum, Battell Block, Middlebury. Program Chairman, Thomas P. Dunleavy, 162 N. Main St., Barre.

Western States Osteopathic Society of Proctology, annual meeting, Westward Ho Hotel, Phoenix, Ariz., October 12-14. Secretary, E. F. Waters, 925 E. South Temple St., Salt Lake City 2, Utah. Program Chairman, Lee R. Borg, 1130 W. Santa Barbara Ave., Los Angeles.

Wisconsin, annual meeting, Schroeder Hotel, Milwaukee, April 12-14. Secretary, Edwin J. Elton, 1518 N. Seventieth St., Wauwatosa 13.

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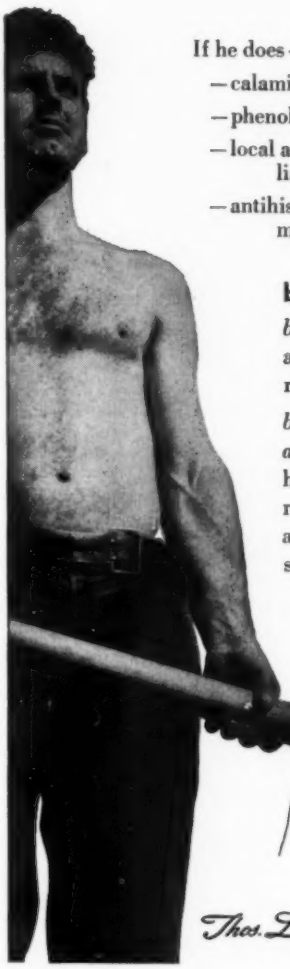

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State and National Boards

ARIZONA

Basic science examinations September 15 at the University of Arizona. Applications must be filed by September 1. Address Herbert D. Rhodes, Ph.D., secretary, Basic Science Board, University of Arizona, Tucson.

COLORADO

Basic science examinations September 9-10, Lecture Room, Y.M.C.A. Bldg., 16th and Lincoln Sts., Denver. Applications must be filed by August 26. Address Esther B. Starks, D.O., secretary, Basic Science Board, 1459 Ogden St., Denver.

CONNECTICUT

Professional examinations November 10. Applications must be filed immediately after candidates have been notified that they have passed the Healing Arts Board. Address Frank Poglitsch, D.O., secretary, Osteopathic Examining Board, 300 Main St., New Britain.

Basic science examinations October 10, Room 23, Lampson Hall, Yale University, New Haven, Conn. Applications must be received no later than 2 weeks before examination. Address Miss M. G. Reynolds, executive assistant, State Board of Healing Arts, 110 Whitney Ave., New Haven 10.

FLORIDA

Basic science examinations November 7 at Gainesville. Applications must be filed by October 26. Address M. W. Emmel, D.V.M., secretary, Board of Examiners in the Basic Sciences, P.O. Box 340, Gainesville.

HAWAII

Examinations October 14. Address Frank O. Gladding, D.O., secretary, Board of Osteopathic Examiners, 504 Hawaiian Trust Bldg., Honolulu 13.

IDAHO

Examinations November 12. D. W. Hughes, D.O., secretary, Board of Osteopathic Examiners, 203 Sun Bldg., Boise.

ILLINOIS

Examinations October 6-8. Address M. C. Hobart Engle, director, Department of Registration and Education, Springfield.

IOWA

Basic science examinations October 13, Capitol Bldg., Des Moines. Applications must be filed prior to examination. Address Ben H. Peterson, Ph.D., secretary, Board of Basic Science Examiners, Coe College, Cedar Rapids.

KANSAS

A. H. Thiemann, Sublette, has been reappointed and Fred E. Dunlap, Pleasanton, has been appointed to the Board

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MAINE

Examinations November 10. Address George F. Noel, D.O., secretary, Board of Osteopathic Examination and Registration, Monument Sq., Dover-Foxcroft.

MARYLAND

Examinations in October. Address Christopher L. Ginn, D.O., secretary, Board of Osteopathic Examiners, 330 N. Charles St., Baltimore 1.

MASSACHUSETTS

Examinations in November. Address Robert C. Cochran, M.D., secretary, Board of Registration in Medicine, Room 33, State House, Boston 33.

MICHIGAN

Basic science examinations October 9-10 at Detroit and Ann Arbor. Applications must be filed by October 1. Address Mrs. Anne Baker, secretary, Board of Examiners in the Basic Sciences, 410 W. Allegan, Lansing.

MINNESOTA

Basic science examinations October 6-7 at Millard Hall, University of Minnesota, Minneapolis. Address Raymond N. Bieter, M.D., secretary, Board of Examiners in the Basic Sciences, 105 Millard Hall, University of Minnesota, Minneapolis 14.

Professional examinations September 5 at Minneapolis. Applications must be received by September 1. Address Wallace F. Kreighbaum, D.O., secretary, Board of Osteopathic Examiners, 2748 Hennepin Ave., Minneapolis 8.

MONTANA

Examinations September 1 at Helena. Applications must be filed 2 weeks in advance. Address Asa Willard, D.O., secretary, Board of Osteopathic Examiners, Wilma Bldg., Missoula.

NEBRASKA

Basic Science examinations October 6-7 at University of Nebraska College of Medicine, Omaha. Applications must be filed by September 18. Address Mr. Husted K. Watson, director, Bureau of Examining Boards, Room 1009, State Capitol Bldg., Lincoln.

NEVADA

Basic science examinations October 6. Applications must be filed by September 24. Address Dr. Frank Richardson, secretary, Board of Examiners in the Basic Sciences, University of Nevada, Reno.

NEW HAMPSHIRE

Examinations September 9-10. Applications must be filed by September 9. Address John S. Wheeler, M.D., secretary, Board of Registration in Medicine, 107 State House, Concord.

NEW JERSEY

Examinations October 20. Applications must be filed 20 days prior to examination. Address E. S. Hallinger, M.D., secretary, Board of Medical Examiners, 28 W. State St., Trenton.

NEW MEXICO

Basic science examinations in October. Address Mrs. Marguerite Cantrell, secretary, Board of Examiners in the Basic Sciences, P.O. Box 1522, Santa Fe.

NEW YORK

Examinations October 6-9 at Albany, Buffalo, Syracuse, and New York City. Applications must be filed at least 30 days prior to examination. Address Stiles D. Ezell, M.D., secretary, Bureau of Professional Examination and Registration, 23 S. Pearl St., Albany 7.

NORTH CAROLINA

Frank R. Heine has been reappointed to the Board of Osteopathic Examination and Registration for a term which will expire in May, 1958.

OREGON

Basic science examinations August 29 at 9 a.m., Room 205, Portland State Extension Center Bldg., 1620 S.W. Park St., Portland. Applications must be filed by August 12. Address Charles D. Byrne, Ph.D., secretary, State Board of Higher Education, Eugene.

RHODE ISLAND

Professional examinations October 1-2. Applications must be filed 30 days prior to examination. Basic science examinations November 18. Address Mr. Thomas B. Casey, Administrator of Professional Regulation, 366 State Office Bldg., Providence.

SOUTH CAROLINA

Examinations November 17. Address Nancy Hoselton, D.O., president, Board of Osteopathic Examiners, 1711 Gervais St., Columbia 1.

WEST VIRGINIA

Examinations October 26-27 at the Greenbrier Hotel, White Sulphur Springs. Applications must be in the secretary's office 30 days before the meeting. Address W. S. Irvin, D.O., secretary, Board of Osteopathy, 202 Broadway, Berkeley Springs, W. Va.

WISCONSIN

Basic science examinations September 19, 8 a.m.-5 p.m., Assembly Chamber, State Capitol, Madison. Applications must be filed by September 11. Address Professor W. H. Barber, secretary, Board of Examiners in the Basic Sciences, 621 Ransom St., Ripon.

WYOMING

Examinations October 5, State Health Department, New State Office Bldg., Cheyenne. Applications must be filed 2 weeks prior to examination. Address Franklin D. Yoder, M.D., secretary, Board of Medical Examiners, New State Office Bldg., Cheyenne.

REREGISTRATION OF OSTEOPATHIC LICENSES

September 1—Nebraska, $1.00. Address George P. Taylor, D.O., secretary, Osteopathic Board of Examiners, 827 Jackson St., Sidney.

September 1—Ohio, $2.00. Address H. M. Platter, M.D., secretary, State Medical Board, 21 W. Broad St., Columbus 15.

October 31—Pennsylvania, $3.00. Address Mrs. Sara H. Longstaff, secretary, Bureau of Professional Licensing, Harrisburg.

November 1—Missouri, $2.00. Address F. C. Hopkins, D.O., secretary-treasurer, State Board of Osteopathic Registration and Examination, 203 South Sixth Street, Hannibal.

EXAMINATION BY NATIONAL BOARD

The National Board of Examiners for Osteopathic Physicians and Surgeons conducts Parts I and II of its examination on the first Thursday and Friday of each May and December at the six approved colleges. Application blanks may be obtained from the secretary or the dean of the college, and the completed application blank, together with a passport photograph and check for the parts to be taken, must be in the secretary's office by the November 1 or April 1 preceding the examination.

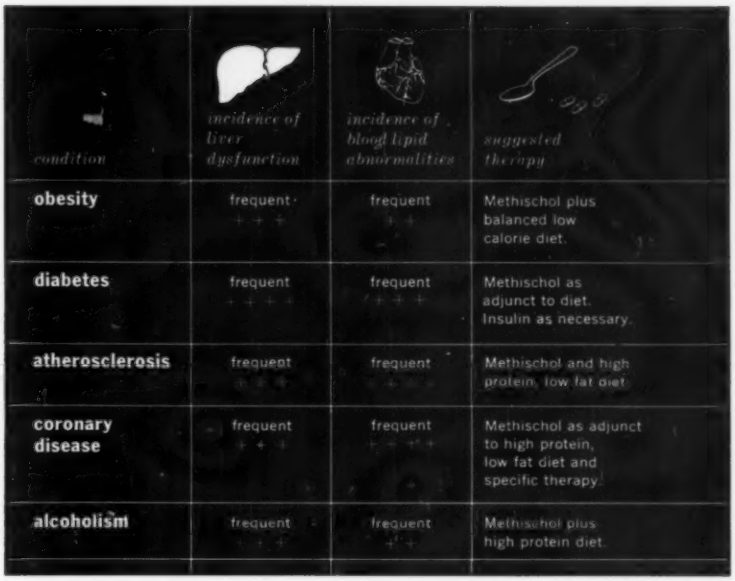
Examinations in Part I consist of anatomy, including histology and embryology; physiology; physiological chemistry; general pathology; and bacteriology, including parasitology and immunology.

Part II consists of examinations in surgery, including applied anatomy, surgical pathology, and surgical specialties; obstetrics and gynecology; pediatrics; neurology and psychiatry; public health, including hygiene; medical jurisprudence; osteopathic principles, therapeutics, including pharmacology and materia medica.

Part III is an oral and practical examination given in Philadelphia, Chicago, Kirksville, and Los Angeles under the supervision of a chief examiner who is a member of the Board and by a panel of associate examiners. Subjects covered in Part III are anatomy; physiology; pathology; osteopathic principles, therapeutics, and pharmacology; surgery; ophthalmology and otorhinolaryngology; obstetrics and gynecology; physical and clinical diagnosis; public health and communicable diseases.

Eligibility requirements are as follows: Part I, satisfactory completion of the first 2 years in an approved school of osteopathy; Part II, satisfactory completion of Part I and of the first two quarters or trimesters of the senior year in an approved osteopathic college; Part III, satisfactory completion of Part II and at least 6 months of a 1 year's internship approved by the American Osteopathic Association. The internship requirement does not apply to candidates who took Part I prior to July, 1950.

Application must be filed with the secretary of the Board not less than 30 days prior to the examination dates. Address Paul van B. Allen, D.O., secretary, 1512 N. Delaware Street, Indianapolis 2, Indiana.



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A PUBLIC RESPONSIBILITY*

By Linwood F. Tice, Ph.G., M.S.†

Pharmacists and physicians in the United States often overlook the unusual but fortunate situation which we enjoy concerning the establishment of drug standards. In most countries a pharmacopeial commission is selected and empowered by the government to choose drugs warranting official recognition and to establish their standards for purity and strength.

In this country, a group of experts, the Committee of Revision of the United States Pharmacopeia, are selected by and from those physicians, pharmacists, chemists, and pharmacologists who represent all organizations having a recognized interest in drug standards. These experts are elected by a democratic process at each Decennial Convention of the United States Pharmacopeia. They, then, serve until their successors are elected at the next convention.

The National Formulary Committee, likewise, is composed of experts selected by the American Pharmaceutical Association without political or governmental interference.

The Congress has recognized the finished work of these two groups—the United States Pharmacopeia and the National Formulary—as establishing the legal standards for drugs in the United States. Supplementing these two books of legal standards are New and Nonofficial Remedies and Accepted Dental Remedies. The former represents those drugs meeting the qualifications as to nature, use, claims, and so forth imposed by the Council on Pharmacy and Chemistry of the American Medical Association. Accepted Dental Remedies lists those meeting the requirements of the Council on Dental Therapeutics of the American Dental Association.

These four books, U.S.P., N.F., N.N.R., and A.D.R., are true examples of democracy in action and ones of which we, as a free people, can rightfully be proud.

As in all democratic institutions, however, freedom imposes with it a responsibility, and those who help establish policy and guide the affairs of these books must keep this great public responsibility constantly in mind.

†Mr. Tice, professor of pharmacy and dean of the Philadelphia College of Pharmacy and Science, acted as panel moderator. Mr. Tice is also a member of the revision committee of the United States Pharmacopeia.
*Reprinted from *Public Health Reports*, May, 1953.

THE NATIONAL FORMULARY*

By Justin L. Powers, Ph.D.†

The present status of the National Formulary stems from the authority derived from the Federal Food, Drug, and Cosmetic Act of 1938. The act states in part that the term "drug" means "articles recognized in the official United States Pharmacopoeia, the official Homeopathic Pharmacopoeia of the United States, or the official National Formulary, or any supplement to any of them . . ."

Section 501 (c) of this statute requires that drugs purporting to be those listed in the National Formulary must conform to the standards of strength, quality, purity, and identity prescribed by that compendium. All determinations of these standards must be made in accordance with the methods described therein. Variations from these standards are permitted only when the identity of a drug is unchanged, and certain labeling requirements of the act are met.

The same section of the act confers authority upon the Administrator of the Food and Drug Administration to prescribe tests where none have been provided or where those described are, in his opinion, insufficient. Before this provision can be invoked, a complicated procedure spelled out in the act must be followed. Not once in 13 years has the Food and Drug Administration found it necessary to invoke this safeguarding provision that gives the right to take away from the National Formulary and the United States Pharmacopeia a part of their standard-making functions. I

*Reprinted from *Public Health Reports*, May, 1953.

†Dr. Powers is chairman of the Committee on National Formulary, American Pharmaceutical Association, Washington, D. C.

think this is a significant point in establishing the role of the National Formulary in maintaining sound drug standards.

Another section of the act declares that a drug shall be deemed misbranded if it purports to be a drug recognized by name in an official compendium unless it is packaged and labeled as prescribed therein.

BASIS OF ADMISSIONS

Admissions of drugs to the National Formulary are based upon their therapeutic value, the extent of their use, or both. When therapeutic value is a consideration, admissions to the 10th edition of the National Formulary will be based on the advice and recommendations of a special committee of medical consultants.

Where extent of use is the principal criterion for admission, it is determined by a consideration of trends in drug therapy, by information obtained from prescription ingredients, by extent-of-use surveys conducted by the National Formulary and others, and by a study of drug market reports.

Prescription surveys which report results only in terms of types of drugs used in compounding and dispensing are of little significance to the Committee on National Formulary of the American Pharmaceutical Association in determining extent of use. These surveys usually report that a large percentage of the prescriptions studied are for "specialties," a small percentage are for U.S.P. drugs, and a still smaller percentage are for N.F. drugs. These reports have the added disadvantage of creating an incorrect impression about the role of the official compendiums in developing and maintaining sound standards for drugs.

Of greater service are surveys such as that by J. S. Mordell, in which the incidences of occurrences of drugs in prescriptions are recorded in terms of official names in addition to the restricted names. We all know that many official drugs are distributed under one or more trade-mark names, but they are still official drugs because in their labeling they purport to be items whose names and standards appear in the official compendiums. We sometimes forget that an official drug by any other name than an official name is still an official drug and must comply with official standards. We are finding the information in Mordell's comprehensive study useful in deciding on deletions from the ninth edition of the National Formulary.

In contrast to the basis of admissions to the National Formulary, the United States Pharmacopeia has followed a more conservative policy. In general, its scope has always been restricted to drugs selected by representatives of the medical profession and believed by them to possess the greatest therapeutic merit. Extensive duplication of drugs having essentially the same action in any single therapeutic classification has been avoided as far as feasible. This selectivity has

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prevented the inclusion of many drugs of therapeutic value or extensive use, or both, and is responsible for the origin and development of the National Formulary.

TWO VALUABLE SERVICES

The National Formulary performs a unique service by providing official standards for extensively used and therapeutically effective drugs not covered by the Pharmacopeia. It may establish specifications for widely used drugs such as rutin or vitamin E concerning the utility of which medical opinion is divided. It provides for the continuance of official standards for drugs deleted from the United States Pharmacopeia during periodic revisions. Many of these drugs continue to be used extensively for many years after losing their Pharmacopeial status. We believe the ultimate consumer is entitled to assurance of the integrity of drugs in these categories through the protection offered by official standards. During periods of shortages of critical materials used in the manufacture of dosage forms, as when glycerin was scarce during World War II, the National Formulary has been instrumental in developing official specifications for safe and satisfactory replacements.

The National Formulary also performs a distinct service to pharmacists and pharmaceutical manufacturers by providing specifications for the procurement of drugs used in dispensing, prescription-compounding, and manufacturing, and in formulas and working directions for the preparation of dosage forms.

We are conscious that the official compendiums ought to be made more useful to the practicing pharmacist. I believe this can be accomplished best by furnishing certain background information which he can use in his everyday practice. Plans have been formulated for doing this in subsequent revisions of the National Formulary by the device of an appendix, the content of which cannot be construed as constituting official standards.

APPLICATIONS FOR MEMBERSHIP

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Adams, Everett H., (Renewal) 77 S. Main St., West Hartford 7

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CHANGES OF ADDRESS AND NEW LOCATIONS

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Bailes, Oscar J., from Kansas City, Mo., to 1122 Mercer St., Princeton, W. Va.
Baker, William F., from 4413 River Oaks Blvd., to Route 11, Box 744, Fort Worth, Texas
Bordwell, Alden S., from 223 Loring Bldg., to 4075 Seventh St., Riverside, Calif.
Braunschweig, Henry J., from 1532 E. Grand Ave., to 1836 E. University St., Des Moines 16, Iowa
Briscoe, Richard W., from 4920 Mansfield Highway, to 2516 Mansfield Highway, Fort Worth 5, Texas
Brom, Harry J., from Dallas, Texas, to 117 S. Pecan St., Arlington, Texas
Brown, J. Francis, from 801 W. Tenth St., to Osteopathic Bldg., 1608 Washington, Amarillo, Texas
Buckalew, Edmund B., from 216 W. Main St., to 211 E. Main St., Girard, Pa.
Bueno, Jesse R., from 2126 E. First St., to 2128 E. First St., Los Angeles 33, Calif.
Cain, Richard F., from 414 S. 30th Ave., to 210 Liberty Bldg., Yakima, Wash.
Callahan, John M., from Farmington, N. Mex., to 220 Lemay Ferry Road, Lemay 23, Mo.
Camnitz, Leonard, from Portland, Maine, to 2391 Webb Ave., Bronx 63, N. Y.
Carpenter, Joseph S., from 12714 Market St., to 1412 Ahrens Drive, Houston 17, Texas
Cary, James E., from 205 E. 11th St., to 1502 Berry Road, Houston 16, Texas
Casey, Wilbert B., from Detroit, Mich., to 224 S. Detroit St., Bellefontaine, Ohio
Chase, Alice, from Nanuet, N. Y., to 6 Ridge Ave., Spring Valley, N. Y.
Ciriaco, Samuel Edward, from Reading, Pa., to 1004 El Hatco Drive, Temple, Pa.
Clarke, Robert N., from 917 Grand Ave., to 3353 E. 27th St., Kansas City 1, Mo.
Coan, Paul J., from Des Moines, Iowa, to 515 W. College, Farmington, Mo.
Coles, Theodore K., from 4220 S. Broadway, to 3962 S. Logan, Englewood, Colo.
Corbett, H. Raymond, from Hinton, Okla., to Velma, Okla.
Cross, Gertrude E., from 2392 E. Main St., to 2690 E. Main St., Columbus 9, Ohio
Cross, Robert B., from 2392 E. Main St., to 2690 E. Main St., Columbus 9, Ohio
Darling, W. E., from Detroit, Mich., to 52 Louise Ave., Highland Park 3, Mich.
DeShong, D. Alfred, from 1202 Third St., to 1802 Morgan, Corpus Christi, Texas
Dickerson, Gerald W., from Kansas City, Mo., to Jackson, Mo.
Dickson, James H., from Craig, Colo., to 7190 W. 38th Ave., Wheat Ridge, Colo.
Donley, Lester W., from Kansas City, Mo., to Donley Osteopathic Hospital, 437 N. Cedar St., Kingman, Kans.
Doron, Chester L., from Cleveland, Ohio, to Pine Lane, Peninsula, Ohio
Eggert, Robert F., from 4701 E. Ninth Ave., to 3125 E. Sixth Ave., Denver 6, Colo.
Ellsworth, Spencer D., from Safford, Ariz., to 101 W. Ajo Way, Tucson, Ariz.
Elston, William L., from Rock Creek, Ohio, to Park Ave., Cortland, Ohio
England, Lloyd L., from Kirksville, Mo., to Craig, Mo.
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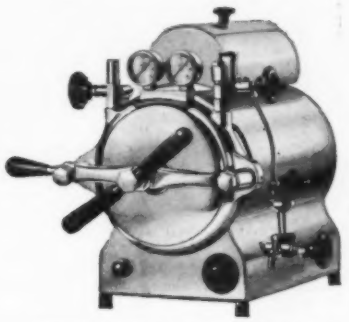

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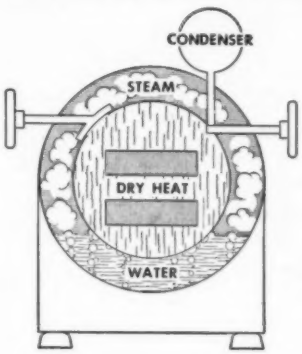

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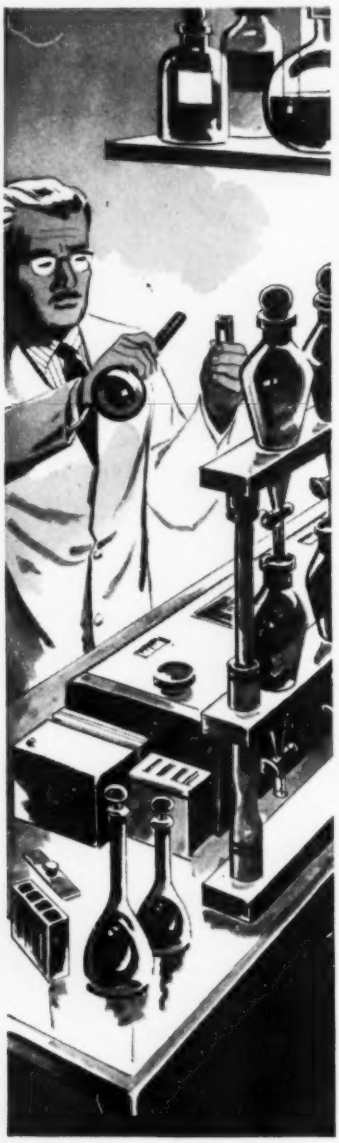

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Fox, David, from Des Moines, Iowa, to 315 Morse St., Camden 5, N. J.
Galutia, Paul R., from Allentown, Pa., to 6 Mitchell Ave., Yonkers 2, N. Y.
Gershon, Julian Robert, from Grand Rapids, Mich., to Clare Clinic, 112 W. Fifth St., Clare, Mich.
Grant, Watson L., from 5115 Montrose Blvd., to 5227 Airline Road, Houston 9, Texas
Greaves, John A., from 8329 Broadway, to 3915 Main St., Kansas City, 2, Mo.
Greenman, Philip E., from Philadelphia, Pa., to 354 Nassau Ave., Kenmore 23, N. Y.
Haffenden, James T., from Grand Rapids, Mich., to Urbandale Clinic, 1331 W. Michigan Ave., Battle Creek, Mich.
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Hampton, Robert G., from San Lorenzo, Calif., to 249 Estudillo Ave., San Leandro, Calif.
Hardy, Lynn W., from 111 N. Second West St., to 1189 E. 27th South St., Salt Lake City 6, Utah
Harkness, Stuart F., from 422 38th St., to 617 Fleming Bldg., Des Moines 9, Iowa
Hawk, Fred E., from O'Fallon, Mo., to 210 Sixth Ave., St. Albans, W. Va.
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Henshaw, Raymond E., from Columbus, Ohio, to Etna, Ohio
Herr, Paul W., from Lancaster, Pa., to Quarryville, Pa.
Herzog, Eugene C., Jr., from Detroit, Mich., to 8564 Lake St., Port Austin, Mich.
Hoffman, Martin J., from 1253 W. Market St., to 1359 N. George St., York, Pa.
Hubach, Walter B., from 14556 Ventura Blvd., to 4166 Dixie Canyon, Sherman Oaks, Calif.
Huffman, Maxwell W., from 620 Bennington Ave., to 5242 St. John Ave., Kansas City 1, Mo.
Hull, James G., from Kansas City, Mo., to 223 Magnolia Ave., Daytona Beach, Fla.
Huneryager, Lloyd W., from Turley, Okla., to Commercial Hotel, Collinsville, Okla.
Hyatt, James E., from Albany, Ore., to 426 Oregon Bldg., Salem, Ore.
Inwood, Garfield, from Chicago, Ill., to 677 Diamond Ave., Canon City, Colo.
Irish, Ralph E., from Scottsdale, Ariz., to Drawer 578, Limon, Colo.
Izbicki, Harry C., from 949 E. 28th St., to 952 E. 31st St., Erie, Pa.
Johnson, Melvin E., from Kansas City, Mo., to 4737 Booth St., Kansas City, 3, Kans.
Karpman, Benjamin, from 127 S. Utah St., to 3509 E. First St., Los Angeles 63, Calif.
Katz, Herman, from 398 Sixth St., to 807 D St., San Bernardino, Calif.
Kenney, Richard R., Jr., from New Paris, Ohio, to Main & Mills, Eldorado, Ohio
Kesler, Guy B., from 312 Orpheum Bldg., to 3145 W. Maple St., Wichita 12, Kansas
Kirk, Norbert Lane, from Muskegon, Mich., to 520 E. Wood St., Brookfield, Mo.
Koplovitz, S. L., from Hornersville, Mo., to 711 N. Second St., Harrisburg, Pa.
Kotsch, Charles, PCO '52; West Side Osteopathic Hospital, 1253 W. Market St., York, Pa.
Kupper, Victor D., from 127 S. Utah St., to 3509 E. First St., Los Angeles 63, Calif.
Larlee, Burleigh L., from 43½ S. Gratiot Ave., to 309 N. Gratiot Ave., Mount Clemens, Mich.
Lewis, Leonard A., from Kirksville, Mo., to 715 Morton St., Paris, Tenn.
Logan, James A., from Menomonee Falls, Wis., to Box 75, Lac du Flambeau, Wis.
MacFarland, Frank J., from Kansas City, Kans., to Waynoka, Okla.
Magliocco, Robert R., from 903 E. Juneau Ave., to 1609 E. Royall Place, Milwaukee 2, Wis.
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Raynesford, J. D., from 617 N. Main St., to 519 N. Ninth St., Garden City, Kans.
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Seeglitz, William A., from 327 Franklin St., to 315 Franklin St., Newton 58, Mass.
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Ulbrich, A. P., from Highland Park, Mich., to 18820 Woodward Ave., Detroit 3, Mich.
Unger, Leo F., from Norristown, Pa., to 621 Avenue Z, Brooklyn 23, N. Y.
Wallace, R. Raymond, from 1008-11 Union Natl. Bank Bldg., to 3715 E. Douglas Ave., Wichita 8, Kans.
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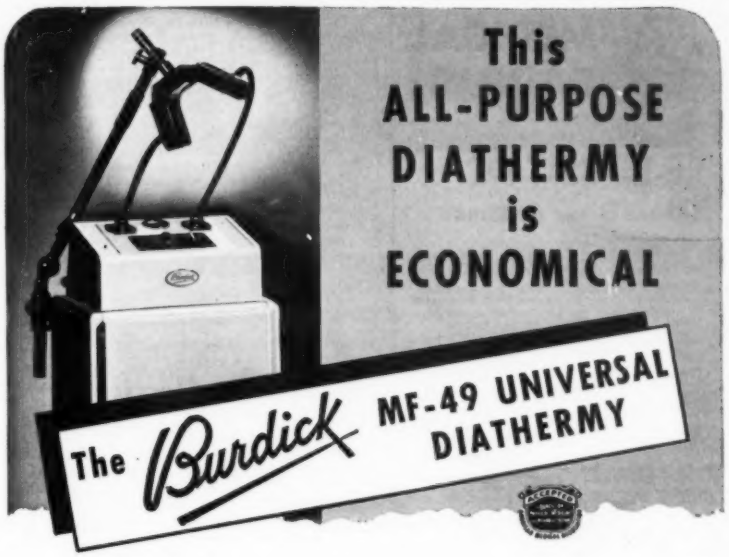

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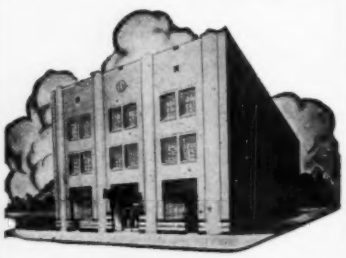

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Williams, Crill M., from 909 Wood Ave., N., to 2001 N. Wood Ave., Linden, N. J.
Willis, Guy E., from La Grange, Ga., to Box 414, Wildwood, Fla.
Wood, William Alexander, from Mount Clemens, Mich., to 7481 Biscayne Blvd., Miami 38, Fla.
Worlock, Harry R., from Ram Bldg., Room 12, to 1427 Park Ave., Aiken, S. C.
Wright, Harry M., from Box 207, to 402 S. Elson St., Kirksville, Mo.
Zachow, Adolphus E., from 320 Illinois Ave., to 322 Illinois Ave., St. Joseph 48, Mo.

Books Received

NUTRITION IN HEALTH AND DISEASE. By Lenna F. Cooper, B.S., M.A., M.H.E., Sc.D., Consulting Dietitian and Formerly Chief of the Department of Nutrition, Montefiore Hospital, New York City; Formerly Food Director, University of Michigan, Dean of School of Home Economics, Battle Creek College; and Supervising Dietitian, U. S. Army, World War I; President, American Dietetic Association, 1937-1938; Edith M. Barber, B.S., M.S., Writer and Consultant, Food and Nutrition; Food Editor, General Features Syndicate; Formerly Food Editor, New York Sun; and Lecturer on History of Cookery and Public Relations for Home Economists, Teachers College, Columbia University; Helen S. Mitchell, A.B., Ph.D., Dean of the School of Home Economics, University of Massachusetts; Formerly Research Professor of Nutrition, University of Massachusetts; Principal Nutritionist, Office of Defense, Health and Welfare Services; and Professor of Physiology and Nutrition, Battle Creek College, and Henderika J. Rynbergen, B.S., M.S., Associate Professor of Science, Cornell University-New York Hospital School of Nursing; Formerly Director of Dietetics, American University Hospital, Beirut, Lebanon; Food Clinic Dietitian, Vanderbilt Clinic, Presbyterian Hospital, New York; Food Clinic Dietitian, Barnes Hospital, St. Louis; and Nutritionist, Community Health Association, Boston. Cloth. Pp. 790. J. B. Lippincott Co., East Washington Sq., Philadelphia 5, 1953.

BALLISTOCARDIOGRAPHY. The Application of the Direct Ballistocardiograph to Clinical Medicine. By William Dock, B.S., M.D., F.A.C.P., Professor of Medicine, State University Medical Center at New York City, College of Medicine; Visiting Physician, Kings County Hospital, Brooklyn; Consultant in Medicine, Veterans Administration Hospital at Brooklyn; Harry Mandelbaum, M.D., F.A.C.P., Lecturer, State University Medical Center at New York City, College of Medicine; Associate in Medicine, The Jewish Hospital of Brooklyn; Physician in Charge, Hypertension and Nephritis Clinic at the Jewish Hospital of Brooklyn; Attending in Medicine, The Jewish Sanitarium and Hospital for Chronic Diseases, Brooklyn; Attending in Medicine, Brooklyn Hebrew Home and Hospital for the Aged, Brooklyn, and Robert A. Mandelbaum, B.A., M.D., Assistant in Medicine, The Jewish Hospital of Brooklyn; Adjunct in Medicine, The Jewish Sanitarium and Hospital for Chronic Diseases, Brooklyn; Assistant in Cardiology, Beth-El Hospital, Brooklyn. Cloth. Pp. 293. Price $9.50. The C. V. Mosby Co., 3207 Washington Blvd., St. Louis 3, 1953.

SURGERY OF THE PANCREAS. By Richard B. Cattell, M.D., Surgeon, The Lahey Clinic; New England Baptist Hospital; New England Deaconess Hospital, and Kenneth W. Warren, M.D., Surgeon, The Lahey Clinic; New England Baptist Hospital; New England Deaconess Hospital. Cloth. Pp. 374, with illustrations. Price $10.00. W. B. Saunders Company, West Washington Sq., Philadelphia, 1953.

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ESSENTIAL UROLOGY. By Fletcher H. Colby, M.D., Chief of the Urological Service, Massachusetts General Hospital; Associate Clinical Professor of Genito-Urinary Surgery, Harvard Medical School, Boston, Massachusetts; Urological Consultant, Lakeville State Sanatorium, Middleboro, Massachusetts. Cloth. Pp. 650, with illustrations. Price $8.00. The Williams & Wilkins Co., Mt. Royal & Guilford Aves., Baltimore, 1953.

THE SURGERY OF INFANCY AND CHILDHOOD. Its Principles and Techniques. By Robert E. Gross, M.D., D.Sc., William E. Ladd Professor of Children's Surgery, The Harvard Medical School; Chief of Surgical Service, The Children's Hospital, Boston. Cloth. Pp. 1000, with illustrations. Price $16.00. W. B. Saunders Co., West Washington Sq., Philadelphia, 1953.

MAN'S BACK. By Theodore A. Willis, M.D., F.A.C.S., Formerly Head, Department of Orthopedic Surgery, St. Luke's Hospital; Consulting Orthopedic Surgeon, U.S.P.H.S. Hospital, Cleveland, Ohio; Member, The American Orthopedic Association; The American Academy of Orthopedic Surgeons; The Clinical Orthopedic Society; Societe Internationale de Chirurgie Orthopedique et de Traumatologie. Cloth. Pp. 161, with illustrations. Price $9.50. Charles C Thomas, Publisher, 301-327 East Lawrence Ave., Springfield, Ill., 1953.

RHEUMATIC FEVER. A symposium held at the University of Minnesota on November 29, 30 and December 1, 1951, under the sponsorship of the Minnesota Heart Association. Edited by Lewis Thomas, M.D. Cloth. Pp. 349. Price $10.00. University of Minnesota Press, Minneapolis 14, 1952.

DISEASE OF THE LIVER, GALLBLADDER AND BILE DUCTS. By S. S. Lichtman, M.D., F.A.C.P., Assistant Professor of Clinical Medicine, Cornell University Medical College; Assistant Attending Physician, New York Hospital; Adjunct Physician, Mt. Sinai Hospital; Assistant in Post-Graduate Medical Instructions, University Extension, Columbia University, New York. Cloth. Pp. 1315, two volumes. Price $22.00 per set. Lea & Febiger, Washington Sq., Philadelphia, 1953.

CORNELL CONFERENCES ON THERAPY. Edited by Harry Gold, M.D. Cloth. Pp. 287. Price $4.00. The Macmillan Co., 60 Fifth Ave., New York 11, 1953.

ESSENTIALS OF MEDICAL RESEARCH. By Wallace Marshall, M.D. Cloth. Pp. 176. Price $3.00. Vantage Press, 120 West 31st St., New York, 1953.

THE USE OF DRUGS. A Textbook of Pharmacology and Therapeutics for Nurses. By Walter Modell, M.D., Assistant Professor, Cornell University Medical College and Doris J. Place, R.N., Instructor in Medical Nursing, Cornell University New York Hospital School of Nursing. Cloth. Pp. 468. Price $4.50. Springer Publishing Co., 1 Madison Ave., New York 10, 1953.

TWENTY-FIVE YEARS OF SEX RESEARCH. History of the National Research Council Committee for Research in Problems of Sex. By Sophie D. Aberle, Member of the National Science Board of the National Science Foundation, and George W. Corner, Carnegie Institution of Washington. Paper. Pp. 248. Price $4.00. W. B. Saunders Co., West Washington Sq., Philadelphia, 1953.

EFFECTIVE INHALATION THERAPY. By Edwin Rayner Levine, M.D., Chairman of the Committee on Physiologic Therapy; American College of Chest Physicians; Former Director of the Chest Service, Michael Reese Hospital, Chicago. Cloth. Pp. 157. Price $4.50. National Cylinder Gas Co., Medical Division, Chicago, 1953.

BACKACHE, BIRTH AND FIGURE RELIEF BY SELF-REVOLVING HIPBONES. By Wm. Schoenau. Cloth. Pp. 264. Price $1.00. Wm. Schoenau, Publisher, 146 S. Norton Ave., Los Angeles 4, 1951.

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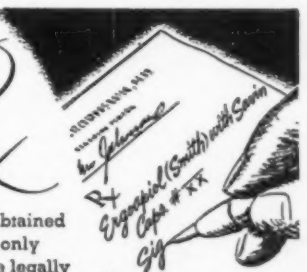

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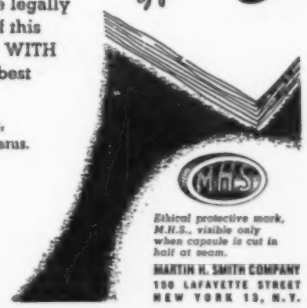

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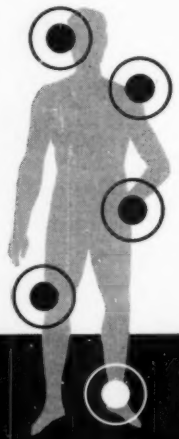

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American Bakers Assn. 9
American Felsol Co. 35
American Osteopathic Association 20, 34, 35, 36
Arnar-Stone Co. 32
Ayerst, McKenna & Harrison, Ltd. 7

Bell-Craig Co. 18
Borcherdt Malt Extract Co. 29, 31
Bristol-Myers Co. ..... Cover II
Burdick Corp. 33

Carnation Co. 12
Central Pharmacal Co. 36
Chal-Yon Corp. 37
Chatham Pharmaceuticals, Inc. 28
Ciba, Pharmaceutical Products Inc. ..... Cover IV
Columbus Pharmacal Co. 33

Dartell Laboratories 32
F. A. Davis Co. 20
Desitin Chemical Co. 24
Devine Bros. 34

Fellows Med. Mfg. Co. 4
Florida Citrus Commission 21

General Electric Co. 15
Gerber's 23
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Holland-Rantos Co. 8

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Thos. Leeming & Co., Inc. 25, 29

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Philip Morris 38
Pelton & Crane Co. 31
Pet Milk Co. 6
Physician's Drug & Supply Co. 37
Picker X-Ray Corp. 1
Professional Foods 10

Raytheon Mfg. Corp. 11
Riker Lab. 16, 17, 40
Ritter & Co., Inc. 26
Roerig, J. B. & Co. ..... Cover III

Saunders, W. B. Company ..... Cover I
Schoomaker Lab. 34
Shield Laboratories 19
Smith, Martin H., Co. 35
Stuart Company ..... Insert
Sunkist Growers 3

S. J. Tutag Co. 10

U. S. Vitamin Corp. 27
Van Camp Laboratories 39
Vitaminerals, Inc. 14

Wocher, Max & Co. 37
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Riboflavin	80 mcg	Threonine	4.1
Niacin	12 mg	Tryptophan	1.1
Animal Protein Factor (B_{12})	8 mcg	Valine	5.2

per 100 grams

(94 calories per 100 grams, or 26.6 calories per ounce)

ACCEPTED COUNCIL ON FOODS AND NUTRITION AMERICAN MEDICAL ASSOCIATION

Both brands the same high Quality

Van Camp Laboratories
Terminal Island, California

Please send samples and complete information on Strained Tuna Baby Food.

*Name*_____

*Address*_____

in many respects
THE SAFEST among the
potent hypotensives

VERILOID®
a unique alkaloidal extract of Veratrum viride

- lowers blood pressure by vasorelaxation independent of vagomotor effect
- no ganglionic or adrenergic blocking
- no danger of postural hypotension
- cardiac output is not reduced
- no compromise of renal function
- cerebral blood flow not decreased
- tolerance or sensitivity rarely develops
- can be given over long periods without loss of efficacy

now in a new and highly
advantageous dosage form

RAUWILOID + VERILOID®

The addition of Rauwiloid (1 mg.) to Veriloid (3 mg.) in one tablet presents unique advantages. Rauwiloid, a mild hypotensive producing virtually no side actions, leads to desirable moderate bradycardia, a feeling of calm tranquility, and rapid remission of symptoms. It apparently potentiates the powerful hypotensive action of Veriloid, thus making it possible for the patient to obtain striking reduction of blood pressure from lower doses of Veriloid, and with less likelihood of side actions. Average dose, one tablet 3 times daily, ideally after meals, at intervals of not less than 4 hours.

OTHER DOSAGE FORMS OF VERILOID

Veriloid
(plain) is available in 1, 2, and 3 mg. scored slow dissolving tablets.

Veriloid-VP
combines Veriloid 2 mg. and phenobarbital 15 mg. (¼ gr.) per tablet.

Veriloid-VPM
adds mannitol hexanitrate 10 mg. per tablet to the formula of Veriloid-VP.

RIKER LABORATORIES, INC.
8480 Beverly Boulevard, Los Angeles 48, California

IT TAKES BALANCE

Nutritional adequacy does not depend upon income. Even a high standard of living is no assurance of balanced nutrition. VITERRA—containing adequate proportions of Vitamins, Minerals and Trace Elements—is designed for those patients whose inadequately balanced diets do not provide their daily requirements of all essential nutrients.

PRESCRIBE Viterra when you want to assure balanced nutrition for your patients.

Each capsule contains

Vitamin A	5,000 U.S.P. Units	Calcium	213 mg.
Vitamin D	500 U.S.P. Units	Cobalt	0.1 mg.
Vitamin B 12	1 mcg.	Copper	1 mg.
Thiamine Hydrochloride	3 mg.	Iodine	0.15 mg.
Riboflavin	3 mg.	Iron	10 mg.
Pyridoxine Hydrochloride	0.5 mg.	Manganese	1 mg.
Niacinamide	25 mg.	Magnesium	6 mg.
Ascorbic Acid	50 mg.	Molybdenum	0.2 mg.
Calcium Pantothenate	5 mg.	Phosphorus	165 mg.
Mixed Tocopherols (Type IV)	5 mg.	Potassium	5 mg.
		Zinc	1.2 mg.

J. B. ROERIG AND COMPANY • CHICAGO

1 *to escape pollens*

2 alternatives for the hay fever patient

2 *to relieve symptoms*

Pyribenzamine®

hydrochloride
(tripelennamine hydrochloride Ciba)

Once atop Pike's Peak, your hay fever patient can enjoy freedom from pollens. But for patients who must remain in a high-pollen environment, you can institute this effective therapy: one or two Pyribenzamine tablets, 3 or 4 times daily.

Alone and as an adjunct to desensitization, Pyribenzamine has proved effective in relieving hay fever symptoms, as evidenced by thousands of published case reports. On the basis of this evidence, no other antihistamine combines greater clinical benefit with greater freedom from side effects.

For your prescription needs, Pyribenzamine 50 mg. tablets are available in bottles of 100 and 1000 at all pharmacies.

Ciba *Ciba Pharmaceutical Products, Inc., Summit, N. J.*

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